THE
LARYNGOSCOPE

AN INTERNATIONAL MONTHLY JOURNAL
ON DISEASES OF THE

EAR-NOSE-THROAT

FOUNDED IN 1896 BY
DR. M. A. GOLDSTEIN, ST. LOUIS

VOLUME LXVII—January-December, 1957.

DR. THEO. E. WALSH, ST. LOUIS
EDITOR

PUBLISHED BY THE LARYNGOSCOPE
640 SOUTH KINGSHIGHWAY - ST. LOUIS (10), MO., U.S.A.

VOLUME LXVII JANUARY, 1957 NUMBER 1

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PREDICTING HEARING LOSS FOR SPEECH FROM PURE TONE AUDIOGRAMS.

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INTRODUCTION.

In recent years there has been a tendency to interpret handicap due to hearing loss primarily in terms of hearing loss for speech. As a result there have been many attempts to find a suitable way of measuring such hearing loss. Some investigators have attempted to measure this loss directly: determining such factors as the threshold of intelligibility for particular samples of speech and the ability to discriminate between speech sounds heard at levels well above threshold. Other investigators have tried to find a way to translate hearing loss for pure tones into hearing loss for speech. This latter method is of particular interest to industry, since industrial hearing tests are made with pure tone audiometers.

It is the purpose of this paper to present a method using pure tone thresholds to predict hearing loss for a certain kind of speech. The prediction of hearing loss for a particular sample of speech is one step toward an understanding of the pure tone-speech relation. It is not our intention to present

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a means for predicting hearing loss for speech in general. The following discussion of equations for predicting hearing loss for a particular sample of speech must be viewed as a progress report on the overall problem of determining the relation between pure tone tests and speech tests.

Many attempts have been made in recent research to find a measure of hearing ability for speech.[1,2,3,4,5] These studies have established, among other things, the existence of a measurable threshold of intelligibility for different groups of words. We are concerned in this discussion with the particular group known as spondee words. They are bi-syllabic words spoken with the same stress on each syllable: baseball, duckpond, railroad, iceberg, hedgehog, etc. A note of caution should be injected here: it is extremely important to keep in mind that spondee words represent only a small part of the large area of speech for communication. Spondee word lists were selected for a specific and limited purpose—to provide words that are homogeneous with respect to intelligibility so that a consistent measure of threshold of intelligibility can be made.

Other word lists have been designed to test other responses. A measure of the threshold of intelligibility of spondee speech is not a measure of ability to communicate by speech. First of all, communication by speech is carried on, not at threshold levels, but well above; further, the speech sounds contained in the spondee word lists are not fully representative of the speech sounds a man must "understand" to communicate verbally. In studying the relation between pure tone thresholds and spondee speech reception thresholds we have confined ourselves to only a part of the problem. When a fully representative speech sample has been developed, a measure of hearing loss for this sample may approximate a measure of loss of ability to communicate by speech. Until such time, however, we can learn much about the relations of hearing loss for pure tones to hearing loss for speech by studying hearing for spondee speech.

MEASURING SPONDEE SPEECH RECEPTION THRESHOLDS.

The threshold of intelligibility of spondee words is determined easily. Lists of the words have been recorded at

Central Institute for the Deaf. Auditory Test No. W-1 is a constant level recording of 36 spondee words. The level at which these words are presented to a subject can be controlled by an attenuator. In Auditory Test No. W-2 the same 36 words are recorded, not at constant level, but in successive steps of decreasing volume. Each step contains six words. In either of these tests, as the subject listens to the recording, he repeats all the words that are intelligible to him until the point is finally reached at which none of the words in a step are intelligible. The level at which he correctly identified half the words is taken as his threshold of intelligibility for spondee words or, more commonly, his *spondee speech reception threshold.* Zero on this threshold scale is chosen to be the same as the average speech reception threshold determined for a group of normal hearing persons. As in pure tone audiometry, thresholds are reported in decibels above or below the normal hearing threshold. Calibrated speech audiometers with provision for checking and setting the zero are available; when they are not used, the zero speech reception threshold must be determined by testing a group of normal listeners.

If a person has an elevated speech reception threshold he is said to have a *hearing loss for speech.* Hearing loss for speech, as the term is used in the literature of speech and hearing, refers only to the speech reception threshold. If the speech reception threshold is the same as the normal average speech reception threshold, the hearing loss for speech is zero. If the speech reception threshold is elevated 10 db, the hearing loss for speech is 10 db. Because of misinterpretations that might occur, readers are asked to keep in mind the limited and specific meaning of the term "hearing loss for speech".

PREDICTING SPONDEE SPEECH RECEPTION THRESHOLDS FROM PURE TONE THRESHOLDS.

Numerous methods have been proposed for predicting speech reception thresholds from pure tone thresholds. Some investigators suggest as a predicted speech threshold an average of pure tone thresholds measured at frequencies considered important to speech reception (the Three Average method averages thresholds for 500, 1000, 1500 cps or 500, 1000, 2000

cps; the Two Best averages the two lowest of three; the Two Worst averages the two highest of three, etc.). Other investigators have proposed predicting equations that combine, not averages, but instead, *unequally* weighted values of pure tone thresholds. In the discussion that follows we present one such equation, show how it may be used to predict speech reception thresholds, and discuss briefly the manner in which it was derived.

THE PRESENT STUDY.

The purpose of the present study was to develop an equation to predict spondee speech reception thresholds from a combination of measured pure tone thresholds. The equation is based on measurements of hearing of the 20 to 29-year-old males (n=319, right ear only) participating in the 1954 Wisconsin State Fair Hearing Survey.* The measurements are: pure tone audiograms made with a standard audiometer, and spondee speech reception thresholds made with a calibrated speech audiometer using Central Institute for the Deaf recordings of Auditory Test No. W-1.

The different weights for the pure tone thresholds appearing in the equation are determined from, *1.* the correlations of measured pure tone thresholds and measured speech reception thresholds, and *2.* the intercorrelations of the measured pure tone thresholds. Hearing losses measured at all seven frequencies, 500, 1000, 1500, 2000, 3000, 4000, and 6000 cps. are considered in deriving the weights.

Hearing losses at these seven frequencies are not equally important to the reception of speech, however, so the derivation of the weights for the predicting equation includes a process that essentially sorts out the frequencies that do have a statistically significant effect on speech reception. Thresholds measured at only these significant frequencies are used in the final equation. Weights for these thresholds are known as regression weights; the final equation is called a multiple regression equation.

Our best multiple regression equation predicts spondee

* Males were chosen because they showed more hearing loss and a greater spread of hearing loss than did the females.

speech reception thresholds from three pure tone thresholds. The equation states that the predicted spondee speech reception threshold in decibels is equal to the constant 6.92 plus 0.22 times the threshold at 500 cps, plus 0.35 times the threshold at 1000 cps, plus 0.21 times the threshold at 1500 cps. This combination of weightings gives the best linear prediction of hearing loss for speech in the group of 20 to 29-year-old males tested with the W-1 records. The equation is:

$$X'_s = 6.92 + 0.22X_{500}\ 0.35X_{1000} + 0.21X_{1500}$$

where X'_s is the predicted speech reception threshold, and X_{500} etc., are the measured pure tone thresholds. The standard error of estimate of the predicted W-1 spondee speech reception thresholds for the 20 to 29-year-old males is 5.0 db. This means that when measured values of the pure tone thresholds are substituted in the regression equation, one can expect that for two-thirds of the predictions the *predicted* spondee threshold will be within 5.0 db. of the *measured* spondee threshold. The remaining one-third of the predictions will differ from the measured value by more than 5.0 db. The correlation of the predicted values, X'_s, and the measured values of the speech reception threshold is called the multiple correlation coefficient. For this group of predictions for the 20 to 29-year-old males it is 0.91.

This is not the only multiple regression equation that can be developed for predicting spondee speech reception thresholds. Other regression equations with other combinations of pure tone thresholds have been computed from the same basic data*. None of them, however, gives, for this group of men, predictions with as low an error and as high a correlation as does the above equation. An equation based on the 1000 cps threshold alone, for example, is:

$$X'_s = 6.6 + 0.73X_{1000}$$

The correlation between the values of the W-1 speech reception threshold thus predicted and the measured values is 0.89. The standard error of estimate is 5.4 db.

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* Regression weights were obtained with the Doolittle solution of the correlation matrix (6, pp. 441-347).

The equation based on the 500 and 1000 cps thresholds is:

$$X'_s = 7.13 + 0.23X_{500} + 0.54X_{1000}$$

The correlation between the predictions made by this equation and the actual measured values is 0.90. The standard error of estimate is 5.2 db. If the threshold at 2000 cps is included, the equation becomes:

$$X'_s = 6.92 + 0.22X_{500} + 0.47X_{1000} + 0.09X_{2000}$$

The multiple correlation coefficient and the standard error of estimate are not changed by the inclusion of the 2000 cps threshold.

It should be emphasized that the weights computed for regression equations such as these, are influenced by the choice of pure tone thresholds measured. The correlations and intercorrelations would yield different weights if, for example, thresholds were measured at 1250 cps instead of 1500 cps, or if thresholds were not measured at 1500 cps at all.

The multiple regression equation based on the thresholds at 500, 1000, and 1500 cps gives the highest multiple correlation and the lowest standard error of estimate of all the equations discussed. The addition of a term including the threshold at 2000 cps does not change either the multiple correlation coefficient, 0.91, or the standard error of estimate, 5.0 db. The predictive importance of thresholds measured at 1500 cps and below is consistent with our knowledge that most of the acoustic energy contained in spondee words is concentrated in the region of lower frequencies. Different weights would be expected if one were predicting thresholds for samples of speech containing more energy in the high frequencies.

COMPARISON OF MULTIPLE REGRESSION EQUATION AND THREE AVERAGE PREDICTIONS FOR OTHER AGE GROUPS.

The multiple regression equation, derived from the threshold data of 20 to 29-year-old males, was tested on other age groups to determine both how well it would predict hearing loss for speech, and how its predictions compared with those of the Three Average method. Two other age groups were selected

for this test: a random sample (n=40) of the men 30 to 39 years of age, and a random sample (n=40) of the men 60 to 69 years of age. All had participated in the 1954 Wisconsin State Fair Hearing Survey. Speech reception thresholds were predicted both by the multiple regression equation and by the more commonly used method of averaging three pure tone thresholds. The Three Average method was applied to thresholds measured at 500, 1000, and 1500 cps; and at 500, 1000, and 2000 cps.

As a check the mean of the Three Average predictions for the 20 to 29-year-olds was compared with the mean of the measured speech reception thresholds. A discrepancy of several db was found: the predicted thresholds were consistently lower than the measured thresholds. The reason for this discrepancy is not clear. It may be due in part to the fact that the speech reception thresholds were measured, not above a normal hearing zero threshold determined at the Survey, but rather above zero on a calibrated speech audiometer. This zero was determined with trained listeners, and it may be below the zero that would be found with untrained ears. Whatever the reason, the mean of the Three Averages is considerably lower than the mean of the speech reception thresholds measused at the 1954 Wisconsin State Fair, and all the Three Averages considered in this paper have been adjusted by an additive constant to compensate for the difference. The constant, determined from the data of the 20 to 29-year-old group, is equal in magnitude to the difference between the mean of the measured speech reception thresholds and the mean of the Three Averages. In other words, a Three Average is used to predict the speech reception threshold in this way:

$$X'_s = \text{Constant} + \frac{(X_{500} + X_{1000} + X_{1500})}{3} = \text{Adjusted Three Ave.}$$

$$\text{where Constant} = \text{MEAN}_{\text{measured speech threshold}} - \text{MEAN}_{\text{Three Averages (20 to 29-year-old group)}}$$

For the sample of 30 to 39-year-old males the regression equation (based on the data of the younger group) and the adjusted averages of the 500, 1000, and 1500 cps thresholds

predict approximately the same speech reception thresholds. The average errors are 4.7 db and 4.8 db respectively. The adjusted mean of the three thresholds 500, 1000, and 2000 cps predicts speech reception thresholds with an average error of 5.1 db. Although this last average does not give as good predictions as do the other two methods, the differences are not large. The errors of the different predictions are given in Table I. Both the average error and the standard error of estimate are given:

TABLE I.

Errors in Predicting W-1 Speech Reception Thresholds from Pure-Tone Thresholds for a Randomly Selected Sample of Men 30 to 39 Years-of-Age in the 1954 Wisconsin State Fair Hearing Survey. (Right Ear Only n = 40).

	Method of Estimating Speech Reception Threshold		
	Regression Equation	Adjusted* Mean of 500, 1000 and 1500 cps Thresholds	Adjusted* Mean of 500, 1000 and 2000 cps Thresholds
Av. Error	4.7	4.8	5.1
Std. Error of Estimate	6.3	6.5	7.7

*Means were adjusted by an amount equal to the difference (for the 20 to 29-year-old males) between the mean of the measured W-1 spondee speech reception thresholds and the mean of the indicated Three Averages.

Similar predictions of speech reception thresholds from pure tone thresholds were made for 40 men whose records were picked at random from the group of 60 to 69-year-old males. Here again, the results obtained with the three methods are not very different. In this particular sample of older men, the adjusted mean of the 500, 1000, and 1500 cps thresholds gave the best predictions of the speech reception threshold. The errors of the prediction for this group are given in Table II:

When the regression equation was developed, it was hoped that it could be used for all age groups. It is apparent, however, that if we are to make the best possible predictions of hearing loss for speech from pure tone thresh-

olds, a different regression equation will have to be prepared for each age group.

INDUSTRIAL AUDIOMETRY AND REGRESSION EQUATIONS.

The purpose of industrial hearing tests is threefold: *1.* to record the status of the worker's hearing at the time of initial employment or reassignment within the company; *2.* to follow the progress of any change in the worker's hearing status during his employment, and *3.* to record a final hearing test when he leaves the company. In our opinion, pure tone tests are absolutely essential for in-

TABLE II.

Errors in Predicting W-1 Speech Reception Thresholds from Pure-Tone Thresholds for a Randomly Selected Sample of Men 60 to 69-Years-of-Age in the 1954 Wisconsin State Fair Hearing Survey.
(Right Ear Only n = 40).

	Method of Estimating Speech Reception Threshold		
	Regression Equation	Adjusted* Mean of 500, 1000 and 1500 cps Thresholds	Ajusted* Mean of 500, 1000 and 2000 cps Thresholds
Av. Error	7.2	6.2	7.6
Std. Error of Estimate	10.3	9.2	10.5

*Means were adjusted by an amount equal to the difference (for the 20 to 29-year-old males) between the mean of the measured W-1 spondee speech reception thresholds and the mean of the indicated Three Averages.

dustrial use. Speech tests do not evaluate thresholds for discrete frequencies within the audible spectrum. The ear is most susceptible to noise-induced hearing loss at 3,000, 4,000 and 6,000 cycles per second, but these frequencies have no significant relation to the hearing and understanding of speech. Speech tests, therefore, cannot be substituted for pure tone tests. A measure of hearing loss for speech may be required, however, for compensation cases. Obviously, tests to measure hearing loss for speech could be provided for use in industry, but in the interests of economy and practicality, a valid method of *predicting* hearing loss for speech from measured pure tone thresholds would be highly preferable.

At present no such valid method of prediction is available. This discussion has repeatedly called attention to the limited speech sample that was used, and to the fact that a measure of the threshold of intelligibility of spondee words is not a measure of the capacity to communicate by speech. There are many parameters that must be investigated before we can formulate a scale by which to assign differences in ability to communicate by speech. One of the first things to be accomplished is the preparation of a representative speech sample for determining loss of intelligibility for everyday speech. If we assume that vowels are related to the sound power of speech, and consonants are related to the discrimination or understanding of speech, our sample should represent both of these speech components; but in what proportion it is still difficult to say. Once the sample is prepared and tested, multiple regression weights for pure tone thresholds can be determined, giving an equation that may be used to predict hearing loss for this representative sample of everyday speech.

The regression weights given in this paper for predicting hearing loss for spondee words, and the regression weights given by Harris[6] for predicting hearing loss for another group of words, (the phonetically balanced list, known as PB's) place an emphasis on pure tone thresholds between 500 and 2000 cps inclusive. Thresholds measured at frequencies below or above these have no significant influence on the predicted values of hearing loss for PB or spondee speech samples. These observations are in complete agreement with the recommendation that 500, 1000, and 2000 cps adequately represent the "speech" frequencies in calculations of speech disability due to hearing loss.

COMPUTATION OF THE MULTIPLE REGRESSION WEIGHTS. ADEQUACY OF THE SAMPLE.

When the weights for the multiple regression equation were computed, the audiometric data were arranged to show both the range and the distribution of hearing loss in the 20 to 29-year-old group. Table III shows these losses at

several percentile points for each frequency and for speech. It can be seen, for example, that 90 per cent of the sample had hearing losses of 29.6 db or less at 2000 cps, 50 per cent had losses of 18.8 db or less at 6000 cps, and 10 per cent had losses of 2.9 db or less when tested with the W-1 spondee word list. To insure that a multiple regression equation will be useful in predicting speech reception thresholds generally, the weights must be computed from data typical of a wide range of hearing loss. Note, in the

TABLE III.

Distribution and Range of Hearing Losses As a Function of Frequency for Men 20 to 29-Years-of-Age in the 1954 Wisconsin State Fair Hearing Survey.
(Right Ear Only n = 319)

Freq.	Hearing Loss at Centile Points (in db) 10th Percentile	25th Percentile	50th Percentile	75th Percentile	90th Percentile	Range of Meas. Hear. Loss db
500	-7.2*	-4.3*	0.5	6.7	16.4	-10 to 95
1000	-4.6	-1.1	3.3	9.9	22.1	-10 to 90
1500	-4.8	-0.9	3.7	10.2	26.8	-10 to 95
2000	-5.0	-0.9	4.3	12.1	29.6	-10 to 90
3000	-1.8	2.8	7.7	20.5	47.1	-10 to 90
4000	-0.1	5.1	13.3	32.6	54.6	-10 to NR
6000	4.0	9.8	18.8	33.4	55.2	-10 to NR
Speech	2.9	5.8	9.4	13.6	23.5	-5 to 80

*This means that 10% of the men tested had hearing losses at 500 cps of -7.2 db or less, 25% had losses of -4.3 db or less, etc.

last column of Table III, that nearly the entire range of hearing loss that can be measured with an audiometer is found at each test frequency.

CORRELATION MATRIX.

Most of the numbers needed to compute the multiple regression weights are contained in Table IV; namely, the two sets of correlation coefficients. The weight assigned to a particular pure tone threshold will be relatively large if the variability of that threshold alone can account for a large part of the variability in the speech reception

threshold. The first row of Table IV contains the correlations of the speech reception threshold and the pure tone thresholds. The rest of the table shows the correlations of the pure tone thresholds with each other. Of the seven pure tone thresholds measured, the 1000 cps threshold has the highest correlation with W-1 spondee speech reception, 0.89.

MULTIPLE REGRESSION WEIGHTS.

Successive steps in the computation of the weights are represented by the columns of Table V. First, the frequencies at which thresholds were measured are listed in

TABLE IV.

Matrix of Zero Order Correlation Coefficients

	500	1000	1500	2000	3000	4000	6000
Speech	.84	.89	.86	.79	.62	.53	.49
500	—	.87	.81	.76	.55	.48	.47
1000	—	—	.90	.84	.63	.56	.54
1500	—	—	—	.89	.65	.52	.50
2000	—	—	—	—	.70	.57	.53
3000	—	—	—	—	—	.81	.69
4000	—	—	—	—	—	—	.82

Column 1. Second, correlations (labeled r) between the W-1 spondee speech reception thresholds and the pure tone thresholds are repeated (for purposes of comparison) in Column 2. Third, standard partial correlation coefficients (β) are listed in Column 3. They are obtained through the inverse solution of the correlation matrix of Table IV. A discussion of such solutions may be found in Walker and Lev[7], p. 331. From these standard partial correlation coefficients one may determine what fraction of the total variability of the speech reception threshold can be accounted for by the individual variability of each of the measured pure tone thresholds. The percentage values are presented in the fourth column of the Table. Adding them, we find that 83 per cent of the total variability of

the measured spondee speech reception thresholds can be accounted for by the variability in the thresholds for the 500, 1000, and 1500 cps pure tones. Finally, partial correlations (b) of the speech and pure tone thresholds are presented in Column 5. These partials are the correlations obtained when one isolates statistically the individual effects of each pure tone threshold on the speech reception threshold. Note that the threshold at 1000 cps, which has the

TABLE V.

Relations Between Pure Tone Thresholds and Spondee Speech Reception Thresholds Obtained by Recorded Auditory Test W-1 for Men 20 to 29-Years-of-Age in the 1954 Wisconsin State Fair Hearing Survey. (Right Ear Only n = 319)

	Pure Tone Thresholds			
Freq.	Zero Order Correlation With Speech (r)	Standard Partial Correlation With Speech (β)	Percent of Total Variability Accounted for in Speech Reception Thresholds (βr)	Partial Correlation With Speech (b)**
500	.84	.26	22	.22
1000	.89	.42	38	.35
1500	.86	.27	23	.21
2000	.79	*	*	*
3000	.62	*	*	*
4000	.53	*	*	*
6000	.49	*	*	*

*Not significantly greater than zero.

** $b = \beta \frac{\sigma_s}{\sigma_t}$ where: σ_s = standard deviation of measured speech reception thresholds; σ_t = standard deviation of measured pure tone thresholds.

highest correlation with the speech reception thresholds, also has the highest partial correlation. The 1000 cps threshold then, has an influence greater than any other single measured threshold on the reception of spondee words at threshold level. Note that the 2000 cps threshold does not have a significant *partial* correlation with the hearing loss for speech. This means that, in and of itself, the 2000 cps threshold bears little relation to the W-1 spondee speech reception threshold. The

relatively high correlation of the 2000 cps threshold with the speech reception threshold, 0.79, is due only to the high correlation of the threshold at 2000 cps, and the thresholds at 500, 1000, and 1500 cps upon which speech reception is dependent. Thresholds measured at frequencies above 1500 cps have partial correlations with the speech reception threshold not significantly greater than zero.

We are now ready to write the multiple regression equation for predicting spondee speech reception thresholds from measured pure tone thresholds. The general form of the equation is:

$$X'_s = a + b_{500}X_{500} + b_{1000}X_{1000} + b_{1500}X_{1500}$$

Where the *b's* are the partial correlations listed in Column 5 of Table V, *a* is a constant needed to insure that the mean of the predictions will equal the mean of the measured values, and the X's are thresholds as originally defined. Substituting the proper numbers for *a* and the *b's* we get the equation discussed earlier.

$$X'_s = 6.92 + 0.22X_{500} + 0.35X_{1000} + 0.21X_{1500}$$

The multiple correlation coefficient for this group of predictions for the 20 to 29-year-old males is .91. The square of the multiple correlation coefficient, $(0.91)^2 = 0.83$, is the coefficient of multiple determination, which indicates how much of the total variability in the hearing loss for spondee speech can be accounted for by the variability of *all* the thresholds appearing in the equation. That is, 83 per cent of the variability of the speech threshold is accounted for by the variability of three pure tone thresholds.

SUMMARY.

A multiple regression equation has been derived to predict W-1 spondee speech reception thresholds from pure tone thresholds. The equation is based on audiometric data obtained from the 20 to 29-year-old males who participated in the 1954 Wisconsin State Fair Hearing Survey. Pure tone thresholds were measured with standard audiometers. Spon-

dee speech reception thresholds were measurd with a calibrated speech audiometer and Central Institute for the Deaf recorded Auditory Test number W-1.

The equation, based on pure tone thresholds measured at 500, 1000, and 1500 cps is:

$$X'_s = 6.92 + 0.22X_{500} + 0.35X_{1000} + 0.21X_{1500}$$

When the 1500 cps threshold is included in the equation, all of the thresholds for higher frequencies have partial correlations that are not significantly greater than zero. This form of the equation gives the best linear prediction of the hearing loss for speech as measured at the 1954 Wisconsin State Fair Hearing Survey by the W-1 spondee speech records. The standard error of estimate for predictions of hearing loss for speech for the group (n=319) of 20 to 29-year-old males is 5.0 db. For the predictions made for random samples (n=40) chosen from groups of older men, 30 to 39 and 60 to 69 years of age, the standard error of estimate is somewhat larger: 6.3 db and 10.3 db respectively.

Other forms of the regression equation were derived with other combinations of pure tone thresholds. The computed weight of a given pure tone threshold was different for each combination of thresholds considered.

REFERENCES.

1. Fletcher, H.; *Speech and Hearing*, New York, D. Van Nostrand Company, Inc., 1929.

2. Sabine, P. E.: On Estimating and Percentage Loss of Useful Hearing, *Trans. Amer. Acad. Ophthalmol.* 46:179-196, 1942.

3. Fowler, E. P.: A Simple Method of Measuring Percentage of Capacity for Hearing Speech. *Arch. Otolaryngol.*, 36:874-890, 1942.

4. Silverman, R. S.; Hirsh, I. J.: Problems Related to the Use of Speech in Clinical Audiometery. *Ann. Otol., Rhinol. and Laryngol.*, 64:1234-1244, 1955.

5. Principles for Evaluating Hearing Loss: Council on Physical Medicine and Rehabilitation, J.A.M.A., 157:1408-1409, 1955.

6. Harris, J. D.: Personal communication.

7. Walker, H. M.; Lev, J.: *Statistical Inference*, New York, Henry Holt and Company, Inc., 1953.

8. Guilford, J. P.: *Fundamental Statistics in Psychology and Education*, New York, McGraw-Hill Book Company, Inc., 1950.

CORRECTIVE SURGERY OF THE NASAL TIP.*†

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It is generally agreed among surgeons engaged in nasal plastic surgery that satisfactory surgery of the tip of the nose offers dificult and intricate problems of corrective surgery.

Anatomic aspects of the cartilaginous structures of the nose have recently been reviewed (Converse, 1955); other pertinent aspects of the anatomy are also referred to in this discussion.

THE NEED FOR TIP SURGERY IN THE NASAL CORRECTIVE OPERATION.

The nasal framework is exposed in the course of the typical corrective rhinoplasty by elevating the periosteum over the nasal bones, and by a transfixion incision extending along the dorsal border of the septum to the septal angle (the antero-superior angle of the septal cartilage), and downward along the lower or free margin of the septal cartilage.

Following modification of the profile by excision of the dorsal hump, the tip area, formed by the alar cartilages, remains unaffected. The septal angle usually remains prominent and must be trimmed. The septum is shortened by resecting a triangular-shaped segment from the lower portion of the septal cartilage (see Fig. 1-A). It is necessary to modify the shape of the nasal tip in order to align it with the remainder of the modified dorsal profile line. If no surgery is performed on the alar cartilages, and the columella is sutured to the lower margin of the septal cartilage,

*Read by invitation at the Sixtieth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Montreal, Canada, May 15, 1956.

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Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, October 14, 1956.

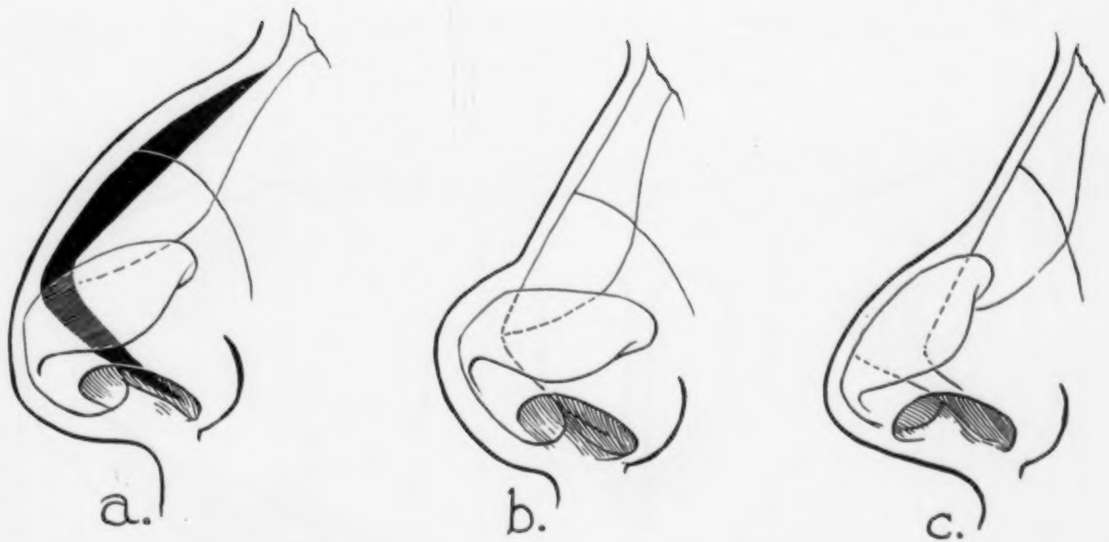


Fig. 1. Illustrating the necessity of breaking the continuity of the alar cartilage in the average nasal plastic operation.
A. The shaded area represents the amount of septal framework excised to correct the shape of the dorsum.
B. Position assumed by the alar cartilages after their approximation to the septal angle.
C. If the continuity of the alar cartilage is not interrupted, the elasticity of the cartilage pulls the tip into the position illustrated.

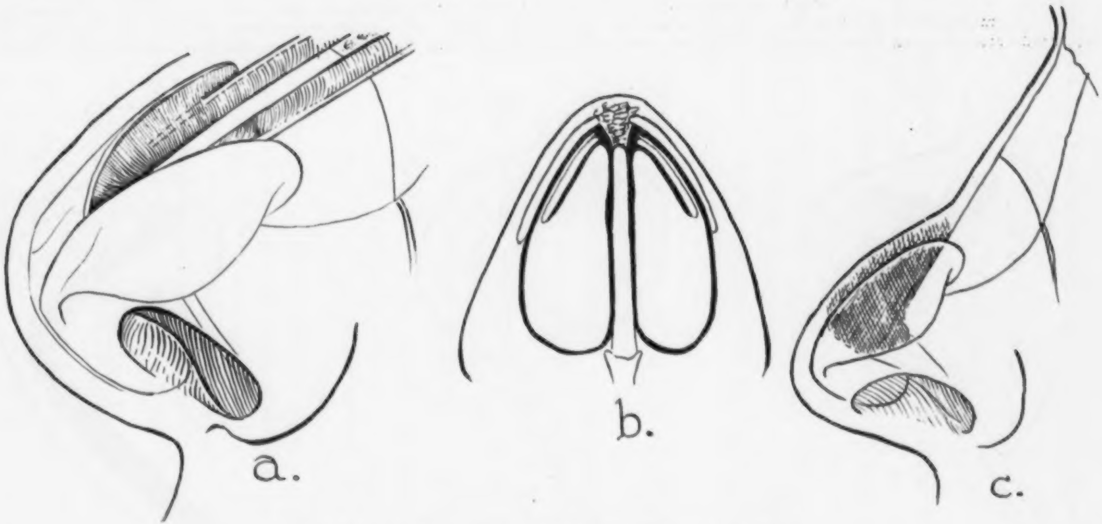


Fig. 2. The subcutaneous pocket in the supra-tip area.
A. Relationship of alar cartilage and septal angle in deformity shown in Fig. 1-C.
B. Frontal section demonstrating the pocket formed between the alar cartilages and the septal angle.
C. The shaded area represents the space between the septal angle and the overlying alar cartilages and skin.

the convexity in the supra-tip area is accentuated, resulting in the typical "parrot beak" profile, and the slight overlap of alar and lateral cartilages becomes accentuated when the tip of the nose is moved upward (see Fig. 1-B). A disappointing condition results if the alar cartilage is permitted to remain in such a position, with the expectancy that the nose will remain shortened. The skin-lined alar cartilages do not adhere to the underlying lateral cartilages. The natural spring of the medial crura eventually results in a downward pull on the tip of the nose, causing a postoperative droop of the tip (see Fig. 1-C). The downward displacement of the tip and the upward displacement of the lateral crura may result in a separation between the septal angle and the supra-tip area (see Fig. 2-A). The septal mucosa joins the vestibular skin on the under surface of the alar cartilage on each side (see Fig. 2-B); the pocket thus formed between the septal angle and supra-tip area causes a characteristic convexity in the area above the tip (see Fig. 2-C).

Three procedures are required in order to prevent such complications: *1*. The alar cartilage must be severed near the junction of the medial and lateral crura to interrupt the continuity of the cartilage and to break its spring-like action; this permits the lateral crus to "lie down" and align itself with the new dorsal profile line. *2*. Shortening of the septum alone does not suffice when the length of the nose is to be reduced; the paired lateral walls must also be shortened by excising a portion of each alar cartilage. This technique is preferable to the resection of large portions of the lateral cartilages, for narrowing of the internal naris and constriction of the nasal airway may ensue. *3*. The alar cartilages should be closely adapted to the septal angle to avoid the formation of a pocket between the cartilaginous structures.

LANDMARKS FOR TIP SURGERY.

The limits of the alar cartilages should be defined prior to surgery. Upward traction on the skin of the dorsum causes blanching of the skin, thus locating the dome of

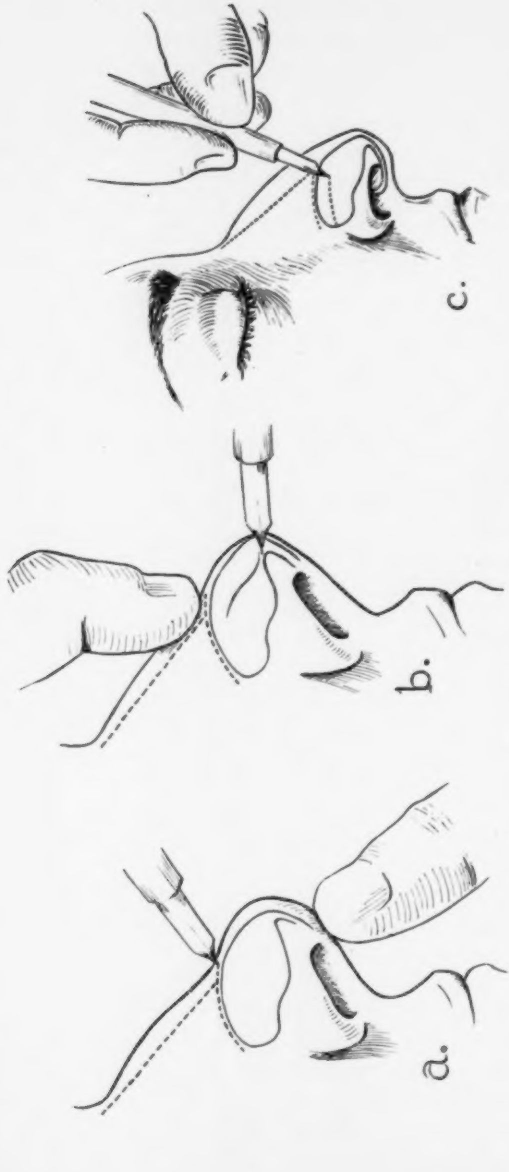


Fig. 3. Landmarks for tip surgery.

A. Outlining the upper border of the lateral crus. B. Marking the apex of the dome. C. Outlining the upper segment of the alar cartilage to be excised.

each alar cartilage (see Fig. 3-A); the tissues resume their normal position when traction on the skin is released. Ink marks are then made on the skin indicating these locations (see Fig. 3-B); the midpoint of the tip is also outlined in ink.

The lower or free margin of the alar cartilage is located by retracting the cartilage and by palpation; the margin is separated from the nostril border by an area of dense collagenous supporting tissue of variable width. A triangular area (the soft triangle) is formed between the border of the naris and the dome by the juxtaposition of lining vestibular skin and covering nasal tip skin. The lateral crus is intimately related to the margin of the nostril immediately lateral to the soft triangle; it then diverges from the margin of the naris. The medial crus is immediately subcutaneous medial to the soft triangle, becoming closely attached to the skin of the columella. Locating the marginal border of the alar cartilage is important, for incisions along this border should avoid the soft triangle; incisions through the soft triangle may result in postoperative notching of the area.

Additional ink lines on the skin outline the bulging upper portion of each lateral crus. These lines, with an additional line marking the upper border of the cartilage, delimit the segment of lateral crus which is to be resected when the nose is shortened (see Fig. 3-C); the width of this segment should approximate the width of the base of the triangle of cartilage which is to be resected from the lower border of the septal cartilage.

TECHNIQUES TO EXPOSE THE ALAR CARTILAGES.

Little faith in the success of purely corrective procedures was expressed by Nélaton and Ombrédanne in their textbook "La rhinoplastie," published in 1904: "The surgeon could not pretend to correct a slight malformation. If a nose be slightly deviated or humped, or show a slight saddle deformity—these are unfortunate defects . . . but we do not believe that the correction of such defects can be achieved

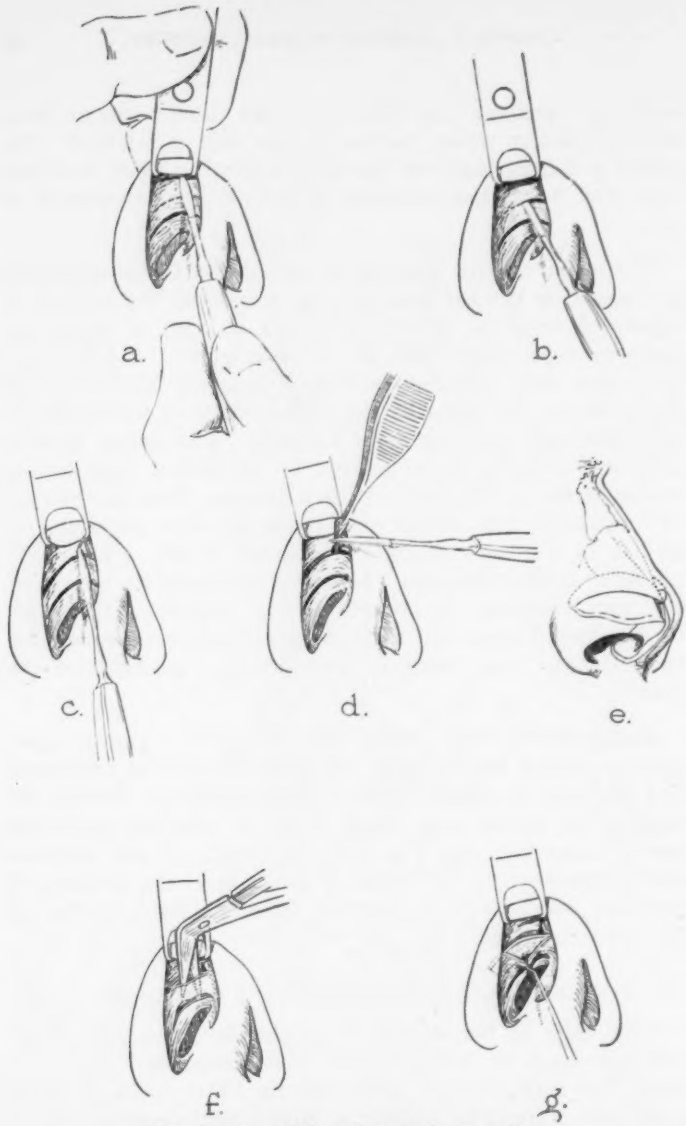
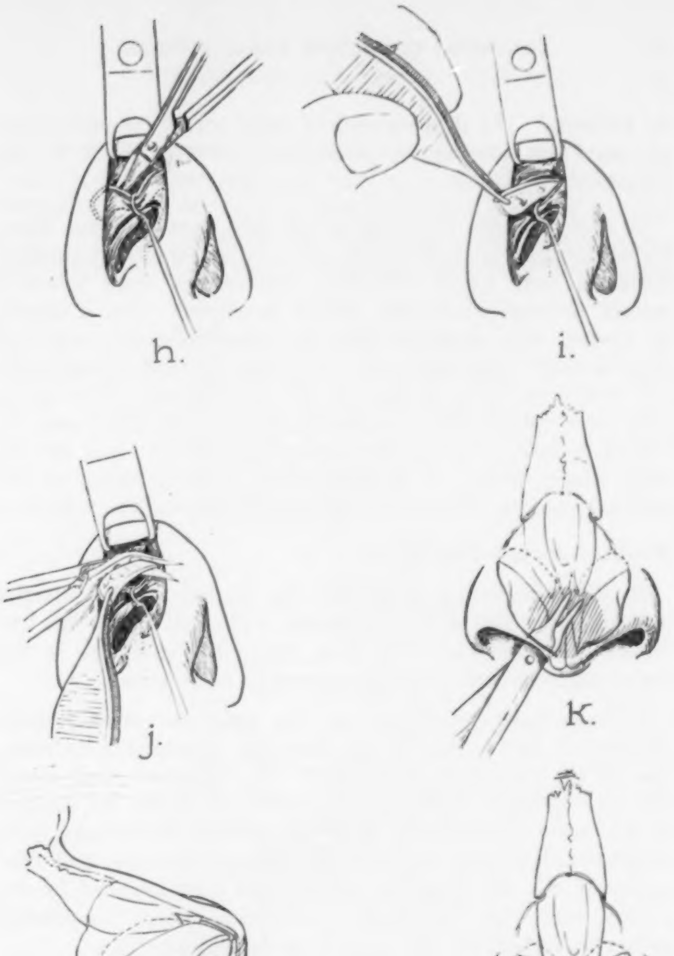


Fig. 4. The modified Joseph tip operation.

A. The incision along the free margin of the alar cartilage. B. The cartilage-splitting incision. C. Incisions outlining small triangle at the dome. D. Excising the triangle. E. Appearance of alar cartilage after excision of triangle. F. Subperichondrial separation of the vestibular lining from the upper cartilaginous segment. G. Upper segment is exposed



by retraction of the vestibular lining. H. Subperichondrial separation of the tissues covering the upper cartilaginous segment. I. Seized with a fine tooth-forceps the lateral end of the upper cartilaginous segment is extruded. J. The upper cartilaginous segment is cut at its junction with the medial crus. K. Subperichondrial raising of the soft tissues over the tip. L. The lateral crus has been mobilized and approximated to the medial crus. M. Frontal view of the cartilaginous structures of the nose at the completion of the operation.

by surgery." The development of nasal corrective operations, of nasal tip surgery in particular, occurred later in the Twentieth Century.

A review of the literature of the last decade of the Nineteenth Century and the early part of the Twentieth Century indicates that nasal corrective procedures were initiated mostly through externally placed incisions. The influence of Joseph, who demonstrated the possibilities of such surgery through internally placed incisions, became a predominant factor in the first quarter of this Century. His teachings were collected in a textbook published in 1931, and the Joseph technique for corrective surgery of the nasal tip has been widely used. A description of a modification of Joseph's technique currently employed by the author follows:

Modified Joseph Technique.

In the preliminary planning, the lateral crus is divided into an upper and a lower segment, indicated by an ink line drawn upon the skin (see Fig. 3-C); the cartilage in the upper segment represents the excess to be removed.

Because block anesthesia of the area has already been attained in the course of the operative procedures, infiltration of the tip is not necessary. The external nasal nerve has been blocked by means of infiltration along the dorsum of the nose; branches of the infra-orbital nerve have been anesthetized by injections at the base of the alae, and filaments of the naso-palatine nerve have been blocked by injection into the membranous septum. Block anesthesia avoids distortion of the tip due to direct injection.

The ala of the nose is retracted, and a small blade knife is used to make two incisions: *1.* An incision along the free margin of the alar cartilage at the dome (see Fig. 4-A); this incision is short but can be lengthened if exposure of the lateral crus is planned; *2.* a cartilage-splitting incision through the vestibular skin and the lateral crus along a line corresponding to the ink line on the skin delineates the limits between the upper and lower segments (see Fig. 3-C and 4-B).

A number of methods can be employed to evaluate the width of the upper segment. A fine needle may be inserted through the ala at the predetermined level (see Fig. 5); the point of perforation of the vestibular skin indicates the intranasal line of incision. Another method consists of de-

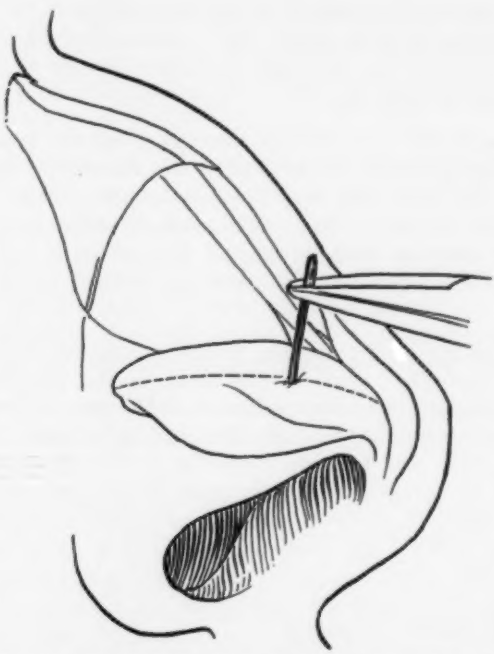


Fig. 5. Useful method to assist the surgeon in placing the cartilage-splitting incision at the correct level. A fine needle is introduced through the skin and cartilage. The point at which it pierces the vestibular lining indicates the position of the cartilage-splitting incision.

termining the relative size of the upper and lower segments on the outer surface of the nose. The free margin of the lateral crus of the alar cartilage is located near the border of the ala, the upper margin of the lateral crus is marked, and the upper segment outlined by an additional line placed at the suitable level; the proportion in size between upper

and lower segments is then determined. These dimensions are relative because of the thickness of the skin overlying the cartilage. The upper segment, for example, may represent one-third of the dimension of the lateral crus. The upper border of the lateral crus is exposed by the intercartilaginous incision. The lower border of the lateral crus is outlined by the incision at the free margin. A third of the distance between these two incisions, beginning at the upper border of the cartilage, indicates the site of the cartilage-splitting incision.

The segments of cartilage resected from the lateral crus are smaller than the areas outlined on the skin. The thickness of the skin and soft tissues extending over the alar cartilages accounts for dimensional differences. The cartilage-splitting incision should follow the curve of the upper border of the lateral crus and extend to the medial crus.

A small piece of cartilage, rather triangular in shape, is excised from the region of the dome in the lower segment before resecting the upper segment of the lateral crus. Two transverse incisions through the vestibular skin and cartilage delimit this triangle (see Fig. 4-C). The triangular segment of cartilage, with its apex at the free margin of the cartilage, is removed with the attached vestibular skin (see Fig. 4-D). The excised tissue serves as an adequate interruption of the continuity of the medial and lateral crura (see Fig. 4-E), and also serves as an approach for additional removal of cartilage from the lower segment, if required.

The next step is the excision of cartilage from the upper segment. The vestibular skin is raised from the cartilage with sharp-pointed scissors (see Fig. 4-F); it is preferable to raise the vestibular skin before separating the cartilage from the covering tissues, for the cartilage becomes mobile after this separation. The vestibular skin is retracted by a hook after being separated from the cartilage (see Fig. 4-G); the assistant controls the retractor and hook, thus freeing the operator. By means of a tooth-forceps in one

hand and angulated scissors in the other, the upper segment of the alar cartilage of vestibular skin is completely freed. The soft tissues over the cartilage are raised subperichondrially with the scissors (see Fig. 4-H). The freed segment of cartilage is severed near its lateral end; if necessary, the lateral portion of the cartilage is also included. The lateral portion of the cartilage is held with the forceps and brought into view (see Fig. 4-I). The cartilage is completely freed medially, is severed at the junction between the lateral and medial crura, and the upper cartilaginous segment is removed (see Fig. 4-J).

Similar procedures are completed on the opposite side of the nose. The remaining portion of the lateral crus is exposed; the incision along the free margin of the alar cartilage is extended laterally, and the lateral crus is freed from the covering soft tissues and brought down with a hook. The upper surface is then exposed and the contour of the cartilage modified. The soft tissues over the medial portions of the domes are elevated subperichondrially (see Fig. 4-K).

A 3-0 plain catgut suture is then placed to join the medial crura and septal angle, and the tip is fitted into the shortened and remodeled nose.

Additional removal of cartilage may be required if bulging is noted along the dorsum. Sharp angles at the cut edges of the cartilage are smoothed. The mobilized structures of the tip are then ready for manipulation; they are placed in a more suitable position (see Fig. 4-L and M) and are retained by adhesive tape placed around and over the tip of the nose.

The Safian Technique.

Safian's technique (1935) is a departure from the Joseph operation. The initial incision extends along the free margin of the alar cartilage from an area immediately medial to the dome along most of the length of the lateral crus of the alar cartilage (see Fig. 6-A and B). Another incision, extended from the medial end of the first one, passes transversely across the alar cartilage, joining with the medial

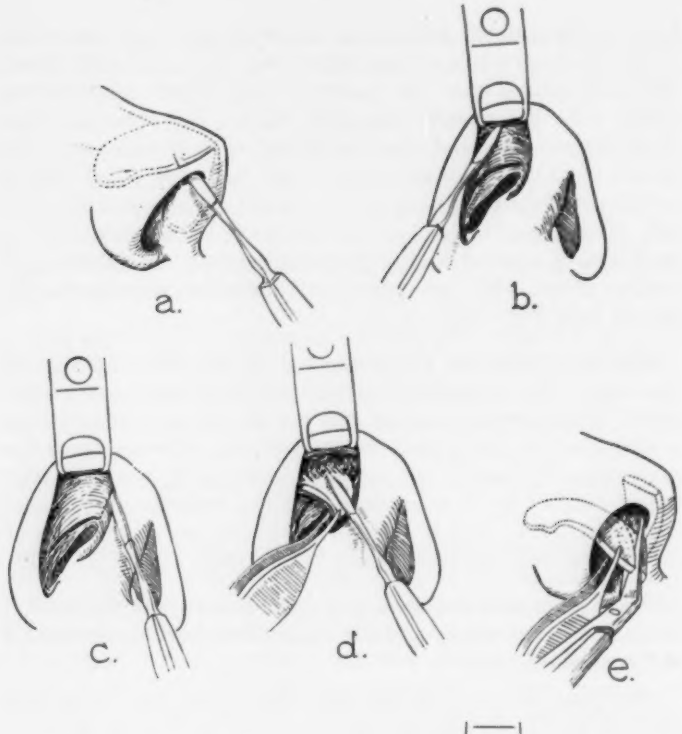


Fig. 6. The Safian Technique.

A. and B. Incision along the free margin of the alar cartilage. C. Incision joining the medial end of the marginal incision to the medial end of the intercartilaginous incision. D. Subperichondrial separation of the cartilage from the soft tissues. E. Trimming the medial border of the cartilage. F. and G. The cartilage is rotated downward and the protruding portion of the cartilage is trimmed.

end of the intercartilaginous incision (see Fig. 6-C); the incision extends medially to the dorsal border of the septal cartilage and includes not only the medial portion of the dome but also all of the medial portion of the lateral crus. Safian and later Fred (1950) have stressed the importance of this part of the technique.

The overlying soft tissues are separated from the outer surface of the cartilage (see Fig. 6-D), and the flap of cartilage and lining vestibular skin is rotated downward and extruded. The vestibular skin retracts along the cut edge of the flap, forming a red margin and exposing a strip of cartilage; excision of this cartilage, along the "red line", is all that is necessary in minor modifications of the tip (see Fig. 6-E). Safian also recommends trimming the lower edge of the cartilage at the dome (see Fig. 6-F and G). This feature of Safian's operation has not been universally accepted because a portion of dome is sacrificed; the cartilaginous excision is, therefore, restricted to the upper and medial portions of the cartilage only. The Safian operation is particularly well-suited in marked hook-deformity of the tip or bulbousness of the nose, due to overdevelopment of the alar cartilages; it is the procedure of choice in secondary corrective operations. The technique offers good exposure of the alar cartilage; the excisions and modifications in contour are made under direct vision.

The Retrograde Technique.

The upper cartilaginous segment is removed from the lateral crus through the intercartilaginous incision in order to avoid a cartilage-splitting incision. Beginning at the intercartilaginous incision, the vestibular lining is elevated from the cartilage by subperichondrial dissection in a retrograde fashion with small angulated scissors; the scissors are then employed to separate the outer surface of the alar cartilage from the soft tissues (see Fig. 7-A and B). An ellipse of cartilage from the upper segment of the alar cartilage is then resected, using a hook to retract the vestibular lining and grasping the upper edge of the alar cartilage with a tooth forceps (see Fig. 7-C).

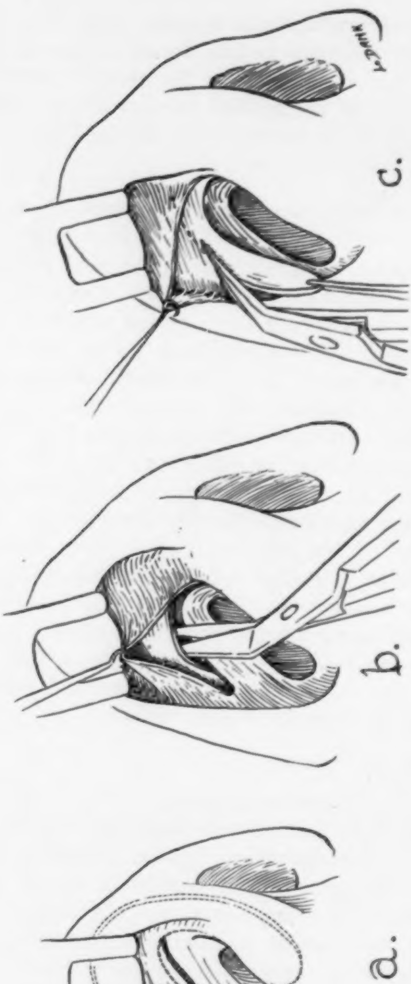


Fig. 7. The retrograde method.

A. Illustrating the intercartilaginous incision for exposure of the nasal framework. B. Using small angulated scissors, the vestibular skin has been raised; the soft tissues are being elevated from the cartilage. C. The upper cartilaginous segment is resected.

The Hockey-Stick Incision Technique.

This curved incision through vestibular lining and lateral crus begins as a cartilage-splitting incision which is then curved forward into the region of the dome (see Fig. 8). A hockey-stick shaped piece of cartilage, which includes the upper segment from the lateral crus and a portion of the dome, can be removed through this one incision.

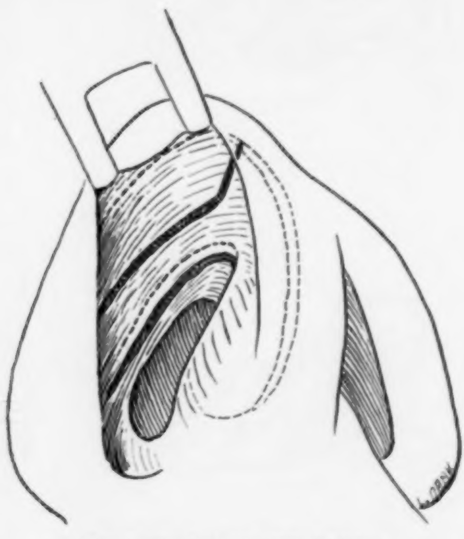


Fig. 8. The hockey-stick incision.
The typical cartilage-splitting incision is extended into the dome by an additional postero-anterior incision.

The Eversion Technique.

This method utilizes the principle of the retrograde technique, and in addition permits eversion of the lateral crus of the alar cartilage, a postero-anterior counter-incision extending through the dome (see Fig. 9-A). It is thus possible to evert lateral and medial crura separately and to perform the cartilaginous excisions without incising the rim (see Fig. 9-C and D).

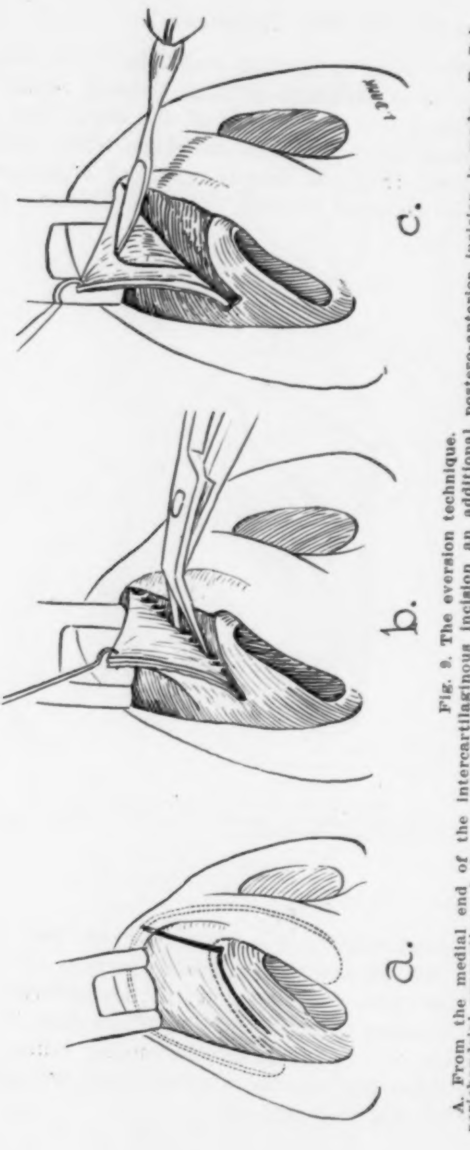


Fig. 9. The eversion technique.

A. From the medial end of the intercartilaginous incision an additional postero-anterior incision is made. B. Subperichondrial separation of the cartilage from the soft tissues. C. The cartilage is everted into an upside-down position and the necessary incisions preparatory to cartilage removal are being made.

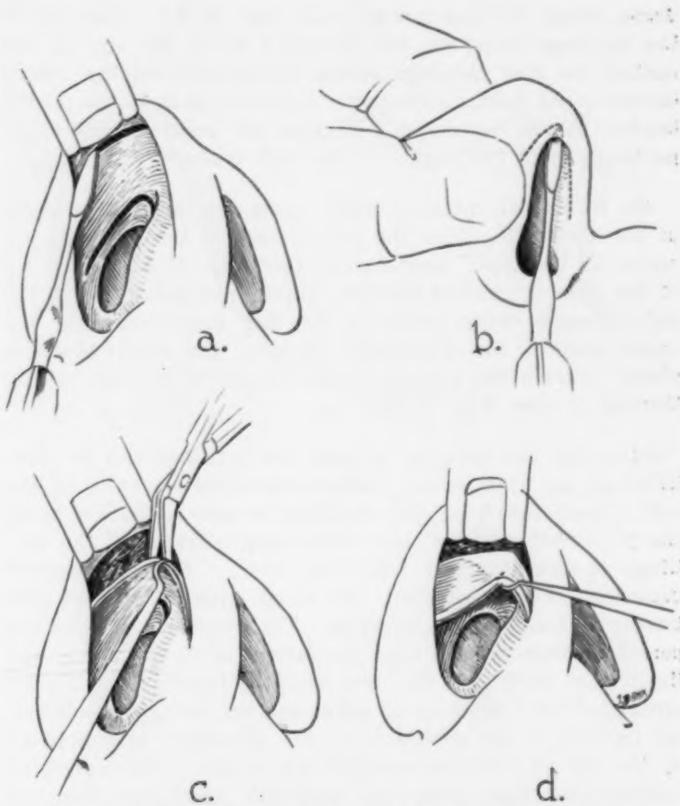


Fig. 10. Technique of complete exposure by rim incision.
A. Incision along the free margin of the alar cartilage. B. Incision along the anterior border of the medial crus. Note position of knife behind the soft triangle. C. Subperichondrial separation of alar cartilage from the soft tissues. D. Traction exerted upon a hook placed under the dome permits extrusion of the cartilage.

Complete Exposure of the Alar Cartilage by Rim Incision.

The ala is retracted, the instrument being placed close to the edge of the nostril. Finger-pressure on the skin surface of the lateral crus causes a protrusion beneath the vestibular skin and thus locates the free margin of the cartilage. An incision is then extended laterally from the

dome, along the free margin (see Fig. 10-A). The rim of the cartilage must be differentiated from the rim of the nostril, the alar cartilage border diverging from the nostril border in its lateral extension. Incisions close to the nostril border should be avoided because of possible distortion, particularly in the region of the soft triangle.

An additional incision, made along the anterior margin of the columella, joins the medial end of the alar rim incision at its upper termination (see Fig. 10-B). The tip of the nose is pinched between thumb and index finger, the soft triangle being puckered by this procedure. At the upper end of the columellar incision, the knife blade is placed beneath the puckered soft triangle to prevent cutting through it (see Fig. 10-B).

After the rim incision is made the cartilage can be identified by its white color. Subperichondrial elevation of the soft tissues overlying the cartilage is now initiated, using sharp pointed scissors; the white shiny surface of the cartilage is thus exposed (see Fig. 10-C). Subperichondrial dissection does not disturb the nasal musculature and soft tissues, and also avoids bleeding. The dissection of adherent perichondrium extends over the lateral crus, the dome, and the medial crus, into the area situated between the medial crura and for a distance of about one cm. along the columellar portion of the medial crus; the procedure is simplified by the use of binocular magnifying loupes. The bipedicled cartilaginous flap, with the attached vestibular skin, is brought down by hook traction (see Fig. 10-D). Upward traction of the alar soft tissues offers additional exposure of the alar cartilage; further modification of the cartilage can then be performed under direct vision. Cartilage may be removed without incision through the lining (Becker 1952), or the cartilage and vestibular lining may be cut through in the region of the dome, as practised by Fomon (1948). The alar cartilage cannot be extruded unless it has been separated from the lateral cartilage by the intercartilaginous incision, and from the septum by the transfixion incision. The intracartilaginous incision should be

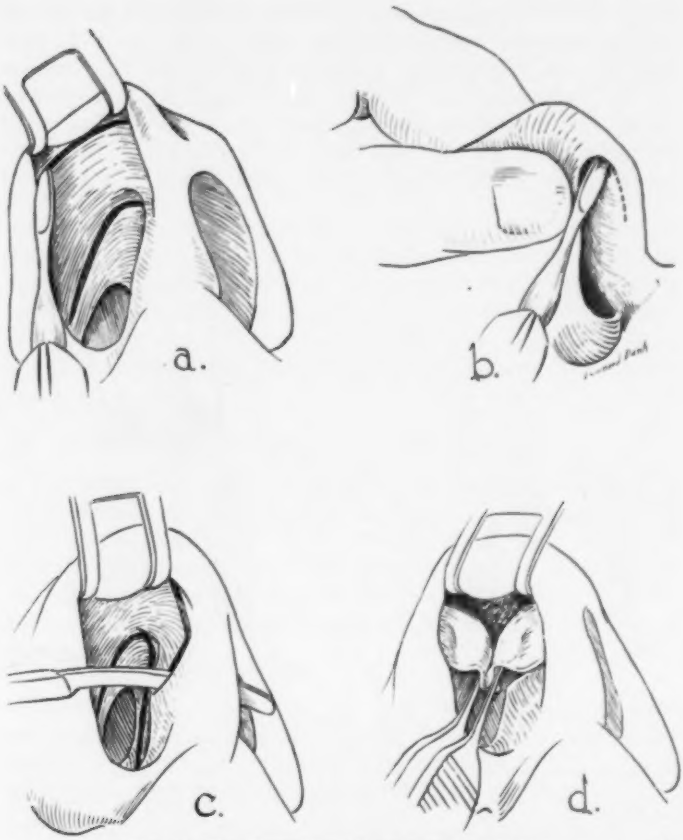


Fig. 11. The Kazanjian tip exposure technique.
A. Marginal incision. B. Incision along the anterior border of the medial crus. C. At the junction of the upper one-third with the lower two-thirds of the columella the transfixion knife severs the medial crura and the columella. D. Both domes may thus be exposed.

long enough to permit adequate mobilization of the lateral crus.

The Kazanjian Exposure.

The initial steps of a technique long employed by Kazanjian are similar to those used for the exposure by rim in-

cision. The marginal incision along the rim of the lateral crus is extended medially to the medial crus (see Fig. 11-A and B). A button-knife is placed through the rim incision on the right side and brought out through the rim incision on the left side after the soft tissues have been raised from the alar cartilage on each side, the knife being placed in front of the medial crura. The medial crura and attached

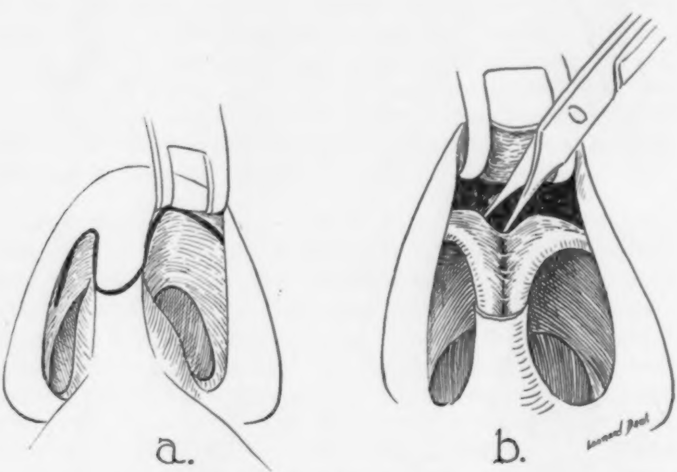


Fig. 12. The Rethi approach.

A. Marginal incisions are made on each side and extended along the anterior border of the medial crus to a point situated at the junction of the upper one-third with the lower two-thirds of the columella. A transverse incision across the columella joins the two incisions. B. The skin of the columella and the tip is raised to expose the area of junction of the domes.

columellar skin are then incised, the incision extending backward and downward and joining with the transfixion incision between septum and columella (see Fig. 11-C). The domes of both alar cartilages and the upper portions of both medial crura are thus separated from the lower portions of the medial crura. By grasping both medial crura with a forceps and retracting the soft tissues of the tip it is possible to extrude both domes (see Fig. 11-D). Sutures approximate the edges of the columellar incision.

The Rethi Exposure.

The technique described by Rethi (1934) employs bilateral alar cartilage rim incisions which extend to the columella and pass along the anterior margins of the medial crura. A transverse incision through the skin of the columella joins the incisions along the margins of the medial crura at the junction of the upper one-third and lower two-thirds of the columella (see Fig. 12-A). The skin over the medial crura is then freed from the cartilages and raised, exposing the area of junction of the domes with the lateral crura (see Fig. 12-B); this technique offers an excellent exposure of the nasal tip. The resultant transverse columellar scar is only slightly visible if the edges of the incision are meticulously sutured.

The Mid-Columellar Vertical Incision.

This vertical incision, frequently employed prior to the development of the intranasal technique by Joseph, affords good exposure of both medial crura and domes. De Kleine (1956) has stressed the value of this incision for exposure in wide and bifid tips because the resultant scar is not visible if correct apposition of the wound edges is obtained.

The Butterfly-Tip Incision.

This incision, advocated by both Kazanjian and New, gives an excellent exposure of the tip. Erich (1953) employed a modification of this incision to correct the tip of deformed noses in cleft-lip patients.

The majority of these techniques provides adequate exposure of various portions of the alar cartilage. Combinations of the techniques have been employed.

There are certain indications for complete exposure of the alar cartilages. These include the wide tips with a tendency to bifidity, and the asymmetrical tips which require surgery of the medial crura. It is not necessary to employ a technique which exposes the entire alar cartilage when the junction of the medial crura is satisfactory in contour. Such a procedure causes disturbance of the anato-

my of the tip and includes the risk of distortion during the healing period. Externally placed incisions should be reserved for complicated tip operations in which the slightly visible resultant scar is of less importance than adequate exposure during operation.

Techniques for Modifying the Contour of the Nasal Tip.

This brief review of methods for obtaining exposure of the tip cartilages also includes descriptions of various techniques for excising cartilage from the upper portion of the lateral crus and the dome. Such cartilaginous excisions are commonly employed in the completion of the surgical correction of the long humped nose, in which the shortening and straightening of the convex profile line are characteristic features. The shape and size of the alar cartilages and their relationship to lateral and septal cartilages present a multiplicity of aspects. The cartilages are thick or thin, and wide or narrow, and show a varied shape in the area of the dome; changes in the shape of the cartilage may be accomplished with the technique of choice. These changes in the past have been brought about primarily by excision of cartilage; too much cartilage has been excised in some cases.

The present trend in tip surgery is to excise less cartilage from either alar cartilage or septal cartilage, for excessive removal of cartilage from the ala leaves it without adequate skeletal support. Contraction of soft tissues during the healing period, occurring without an adequate cartilaginous framework, may result in an irregular appearance of the tip or a pinched appearance and retraction of the nasal border. Excessive removal of septal cartilage results in an open angle between the base of the columella and the base of the alae. The base of the nasal pyramid appears flat, thus increasing the relative distance between the alae; therefore, care should be exercised in planning the amount of cartilage to be removed from the lateral crus, the area of the dome, and the septum.

It may be necessary to excise cartilage in the area of the

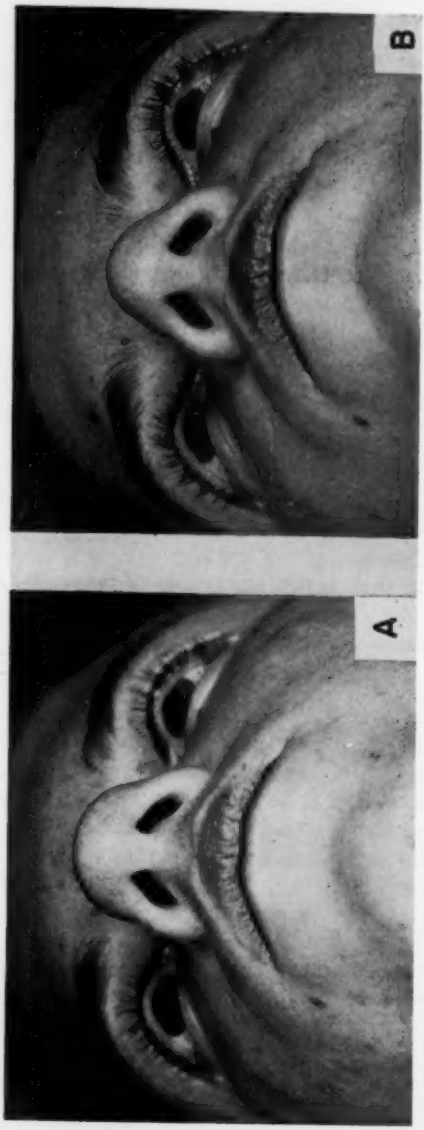


Fig. 13. Reduction in size of broad tip.

A. Preoperative view showing bulbous square-shaped tip. B. Result obtained after adequate excision of cartilage from the area of junction of lateral and medial crura.

dome in order to diminish the transverse dimensions of the nasal tip (see Fig. 13). The domes are in close approximation and shaped with a fairly sharp curve in tips whose contour is considered normal. The wide tip has a wider curve, and the domes are separated in the midline. The contour of the square-shaped tip is determined by the wide flat surface of the cartilaginous domes. Narrowing of such nasal tips requires approximation of the medial crura and excision of a strip of cartilage to narrow the dome (see Fig. 14). Incisions in the region of the dome vary, depending upon the desired modification of the tip. An incision immediately medial to the highest point of the dome is made in tips requiring little modification, except a change in the direction of the lateral crus in order to align the tip to the modified nasal dorsal profile. A triangular segment with its apex below is removed at the expense of the laterally situated cartilage (see Fig. 4-C, D and E). If additional narrowing of the tip is required, the triangle becomes a trapezoid with its wider base above; both elevation and narrowing are produced by cutting through the dome and suturing the medial crura back-to-back as first advocated by Kazanjian (see Fig. 14-A, B, C, and D), or overlapping the lateral crus over the medial crus (see Fig. 14-A, E and F).

Excessive thickness of the lateral crus may be reduced by the excision of thin slices from the surface of the cartilage. Goldman (1953) described a reversed L-shaped incision in the lateral crus to restore normal concavity to this portion of the alar cartilage and to eliminate undue convexity. Some surgeons have cut windows in the alar cartilage to attain similar results; others have felt that it is possible to criss-cross cut the alar cartilage to obtain suitable contour. Cartilage, unlike bone, does not heal to itself by the formation of callus or new cartilaginous tissue; the healing of sectioned cartilage occurs through the intermediary of fibrous tissue alone. The amount of contraction of fibrous tissue is unpredictable and an undesirable displacement of the cut cartilaginous segments may occur. Prudence must, therefore, be exercised in making these incisions.

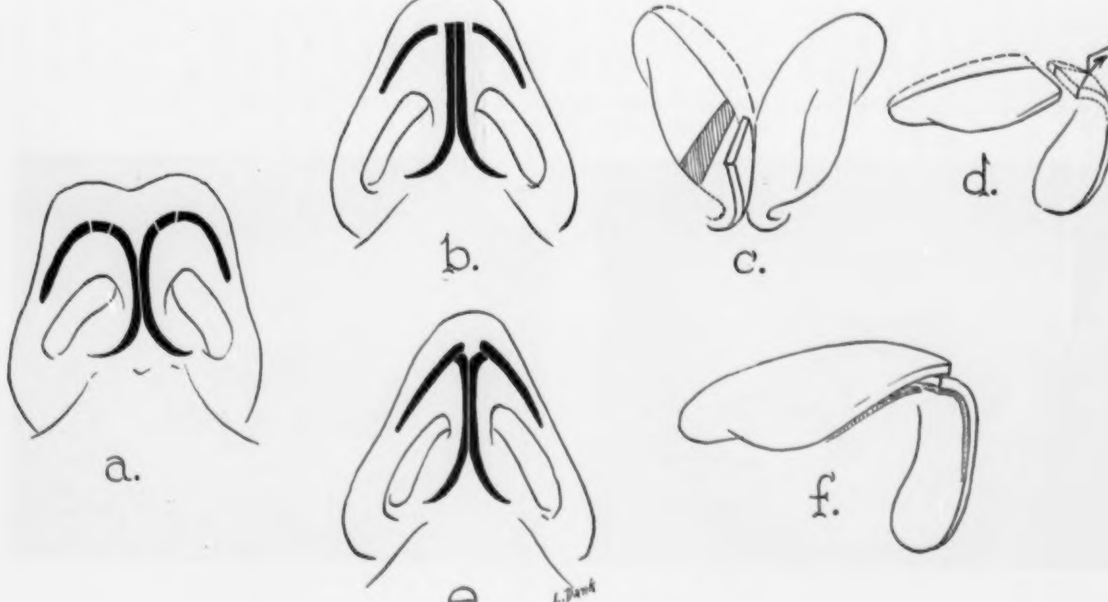


Fig. 14. Schema showing methods for narrowing and raising the tip.

A. In a broad tip excision of cartilage from the domes is required. B., C. and D. Bifidity is corrected and the tip raised by approximating the medial crura. E. and F. Overlap of lateral crus over medial crus narrows and raises the tip. The undersurface of the overlapped lateral crus must be freed of vestibular skin.

Fig. 15. Nasal tip remodelling.

A. Appearance of widened and slightly bifid tip. B. Result obtained by tip remodelling operation illustrated in Fig. 16. Note the natural appearance of the tip.

Three precautions should be noted in surgical modifications of the shape of the alar cartilages: *1. Avoid raw areas resulting from removal of vestibular lining;* the healing of such raw areas results in scar formation and distorting contracture. *2. avoid excessive removal of cartilage which produces a "pinched" appearance;* in the region of the dome excessive removal may leave a gap between the pieces of cartilage resulting in an unsightly groove in the skin of the tip of the nose; *3. sharp angles and protuberances due to overlapping cartilaginous fragments, or improperly shaped cartilaginous implants may produce external irregular prominences.*

A fold of excess skin may occasionally form in the vestibule; this can be trimmed as an office procedure at a later date. Excess vestibular skin never manifests itself on the outer aspect of the nose. The advantages of leaving excess vestibular skin are in marked contrast to the dangers of resecting vestibular skin.

A New Tip-Remodeling Operation.

This operation is based on the principle of remodeling the nasal tip with minimal resection of cartilage and is well-suited for the broad slightly bifid type of nasal tip (see Fig. 15). An adequate amount of cartilage is excised from the upper portion of the lateral crus through a cartilage-splitting incision if the nose requires shortening. The lateral crus, the dome and the upper part of the medial crus on each side are then exposed through a rim incision, as previously described. The connective tissue between the medial crura is removed to permit approximation of the crura. An oblique incision directed superiorly and medially is made through the cartilage of the dome, extending through the cartilage only, and not through the vestibular skin (see Fig. 16-A and B). The point at which this incision is placed depends to a great extent upon the contour of the tip and the amount of narrowing and elevation required. By retracting the skin of the upper portion of the columella to one side it is possible to approximate the media crura with forceps under direct vision, one branch of the forceps be-

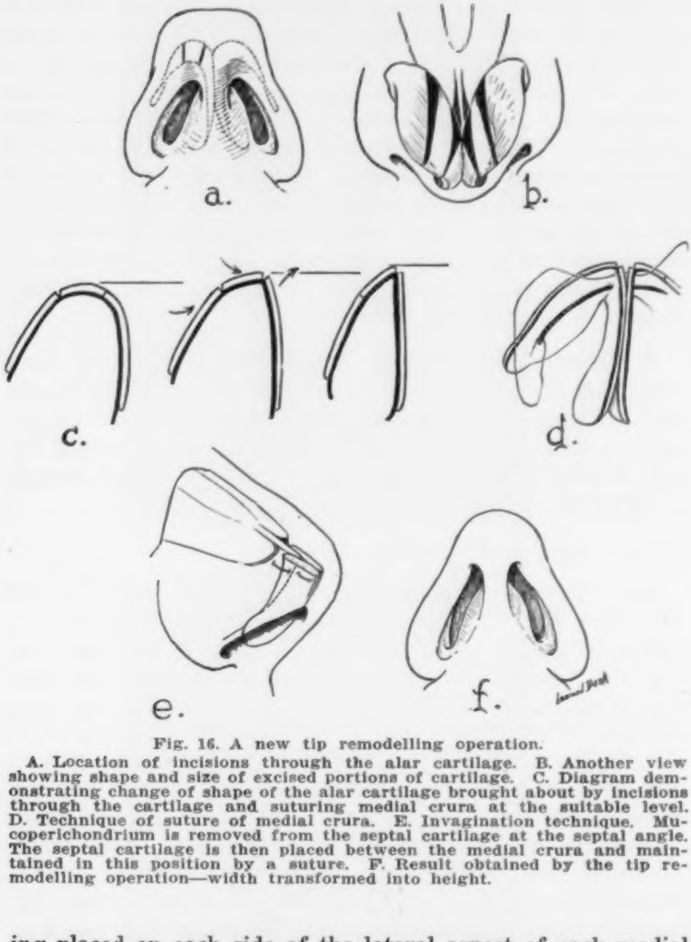


Fig. 16. A new tip remodelling operation.
A. Location of incisions through the alar cartilage. B. Another view showing shape and size of excised portions of cartilage. C. Diagram demonstrating change of shape of the alar cartilage brought about by incisions through the cartilage and suturing medial crura at the suitable level. D. Technique of suture of medial crura. E. Invagination technique. Mucoperichondrium is removed from the septal cartilage at the septal angle. The septal cartilage is then placed between the medial crura and maintained in this position by a suture. F. Result obtained by the tip remodelling operation—width transformed into height.

ing placed on each side of the lateral aspect of each medial crus immediately below the dome. The tip assumes an elevated and narrowed appearance when the domes are thus approximated; maintenance of this position and contour are the goals of the operation.

The location of the oblique cartilaginous incision is de-

termined and the incision is made. The medial crura are approximated more readily because of the break in the continuity of the cartilage (see Fig. 16-C). A convexity or bulging lateral to the dome can be seen. If the tip of the nose is not unusually wide an additional incision at this point will be adequate. In broad tips a strip of cartilage is outlined by parallel incisions and carefully dissected from the vestibular lining. In order to avoid postoperative bulging of the upper portion of the lateral crura, particularly if the nose does not require shortening and the upper portions of the lateral crura are not removed, the excised strips of cartilage should be triangular in shape, with the base of the triangle above (see Fig. 16-B). This procedure removes excess cartilage and permits the lateral crura to "lie down" on the dorsum. If the remainder of the lateral crus still exhibits an undesirable convexity, one or more additional incisions through the cartilage may be employed; these must not extend through the vestibular skin. Three points of the technique should be noted: *1.* The incision at the dome is oblique, thus permitting the greater elevation of the lower portion of the tip, a desirable feature; *2.* the various fragments of cartilage produced by the incisions remain attached to the vestibular skin which is not cut through at any point nor are any cartilaginous fragments even partly separated from the vestibular lining. Because all of the various pieces of cartilage, separated one from the other by incisions, remain attached to vestibular lining, the continuity of the cartilaginous arch is maintained. When the medial crura are sutured together, bringing the domes into the narrowed and elevated position, the various pieces of cartilage follow because of their attachment to the vestibular lining (see Fig. 16-C). *3.* When excising cartilage segments, it should be remembered that these cartilaginous strips are always small, for in the remodeling of the tip width is transformed into height and little cartilage removal is required (see Fig. 16-C and F).

The tip must be adequately suspended to the septal angle before placing the sutures. The nose has been shortened and a portion of cartilage from the free margin of the quad-

rangular cartilage of the septum has usually been excised. The septal angle is denuded of its mucoperichondrium over a distance of a few millimeters to prevent interposition of mucoperichondrium between the cartilaginous structures. Coaptation of the septal angle cartilage to the posterior borders of the medial crura is assured by a correctly placed suture. When postoperative drooping of the tip is anticipated the surface contact between the septal angle and the medial crura may be increased by the procedure described by Rethi (1934) under the name of "embracing flaps", and referred to by Fred (1954) as the "invagination technique". The septal angle, which has been denuded of its mucoperichondrium over a short distance, is thinned by the excision of slices of cartilage from each side if the cartilage is thick. The cartilage is then invaginated between the posterior portions of the medial crura and maintained in this position by a mattress suture (see Fig. 16-E). An additional mattress suture insures coaptation of the lower portions of the columella and the septal cartilage. A mattress suture is placed at the upper portion of the medial crura in order to approximate them in correct position (see Fig. 16-D). This suture is best used with a No. 14 eye-curved cutting needle. The needle is passed into the nose behind the soft triangle in the region of the recess of the vestibule; this suture, which approximates the medial crura one to the other, determines the resulting contour of the tip.

3-0 plain catgut has been found satisfactory and is used throughout for these fixation sutures which do not require removal and do not result in inflammatory reaction. Correct strapping of the tip with adhesive assists in maintaining the position of the cartilaginous segments.

Complete vs. Interrupted Transfixion Incision.

The routine transfixion should be avoided if the nose does not require shortening (see Fig. 17). The complete transfixion procedure results in a degree of downward retraction of the tip due to linear contraction of the healing scar. The transfixion should be interrupted in bulbous nasal tip cases, and in those which do not require shortening; in

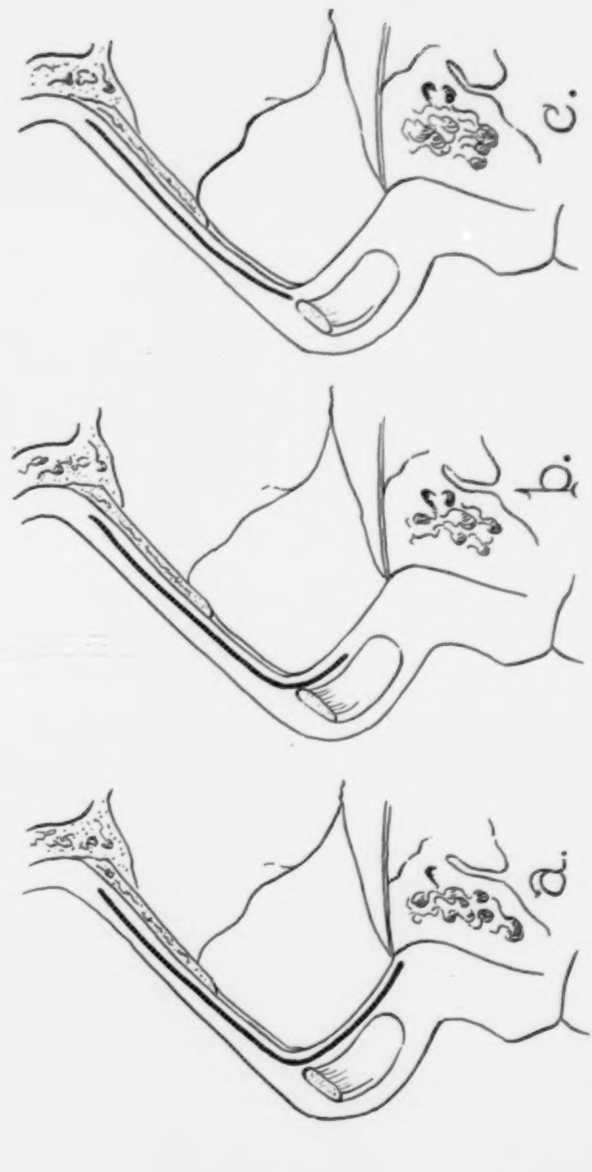


Fig. 17. Complete versus interrupted transfixion.
A. Complete transfixion. B. Transfixion interrupted below the septal angle. C. Transfixion interrupted at the septal angle.

such instances the transfixion incision is stopped at the septal angle (see Fig. 17-C). When slight elevation of the tip is required, the transfixion incision is extended downward around the septal angle for a short distance, sufficient to allow shortening of the septal cartilage in the region of the septal angle (see Fig. 17-B). Adequate exposure is obtained for modification of the dorsal septal profile line, if required.

It is also necessary to extend the incision around the septal angle to permit release of the alar cartilages from their septal attachments when a complete exposure by rim incision is required. In order that the dome and medial crura can be brought down into view they must not be held back by their septal attachment. The intercartilaginous incision should be long enough to permit adequate mobilization of the lateral crus for exposure.

"Raising the Tip."

The term "raising the tip" is employed to signify an increase in the height or projection of the tip; it is also used to designate the change of position of the tip when the nose is shortened. Increase in height or projection of the tip has already been discussed. Raising the tip in conjunction with shortening of the nose is achieved by excising a triangular-shaped piece of septal cartilage from its lower portion. "Drooping of the tip", occurring postoperatively, results from loss of contact between the tip cartilages and the septal angle. The invagination technique, previously described, prevents this drooping. Loss of contact with the septal angle is due to, *1.* overshortening of the septum, thus preventing adequate contact between the cartilaginous structures; *2.* inadequate shortening of the septum: the long nose with its tip pointing downward has not been corrected; *3.* shortening of the septum without shortening of the lateral walls: the downward push of the cartilage causes a downward displacement of the tip; *4.* failure to break the continuity between medial and lateral crura: the elasticity of the alar cartilage causes a recurrence of the original deformity.

A particular problem arises when septal support is lost by trauma or total submucous resection of the septum. The corrected position of the tip must be maintained by shortening the lateral walls through adequate excision of alar cartilage or by overlapping the alar and lateral cartilages. A sufficient amount of vestibular lining is removed from the under surface of the alar cartilage to permit overlap and side to side apposition of the alar and lateral cartilages. After the alar cartilages have been displaced upward, the curvature of the arch formed by the alar cartilages is wider than that formed by the lateral cartilages. Contact is not established unless cartilage is excised from the alar cartilages to diminish the curvature of this arch.

Increasing the Projection of the Tip.

This is a desirable feature in wide tips. When the cartilage has been cut through at the dome, increase in projection can be obtained by overlapping the lateral crura over the dome, and by suturing the medial crura together. The new tip remodeling operation previously described gives satisfactory results in such cases and produces a natural and harmonious nasal tip.

A compromise procedure is required in wide flat tips with thick skin and subcutaneous tissues, or with wide negroid nostrils. Elevating the dorsum of the nose with a bone graft which extends under the tip is a compromise solution (Converse 1955). The lower end of the bone graft extends through the medial crura, a bony columellar strut maintaining the elevation of the nasal tip (see Fig. 18). The inconveniences presented by the rigidity of the nose and the danger of fracture of the bone graft are compensated for by the improvement in the nasal airways and the appearance of the nose. Flatness of the tip may require concommitant lengthening of the columella by a procedure such as the one represented in Fig. 19. This lengthens the columella and diminishes the width between the base of the alae. The defect produced by the mobilization of the lateral flaps is filled by advancing the base of the alae, thus narrowing the nares (see Fig. 20).

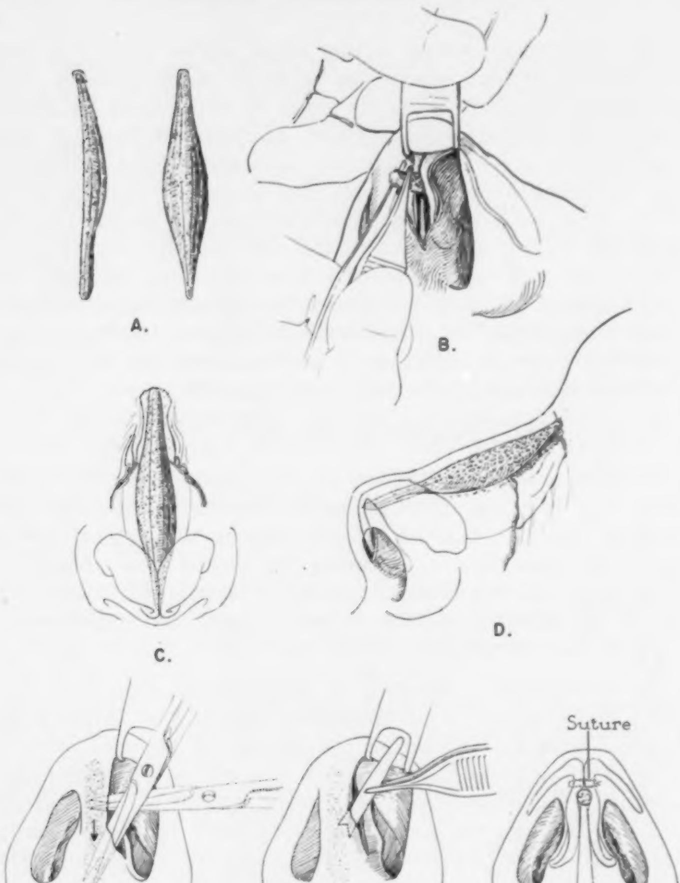


Fig. 18. Raising the nasal tip with supporting bone grafts (From Converse, J. M.: Technique of bone grafting for contour restoration of the face; Plastic and Reconstructive Surgery, 14:332, 1954).

A. Shape of dorsal bone graft. B. Introducing the dorsal bone graft between the medial crura. C. Position of the bone graft over the nasal bones. Subperiosteal contact between bone graft and nasal bones is essential. Note the distal end of the graft beneath the area of junction of the domes of the alar cartilages. D. Profile view of the dorsal bone graft. E. The pocket being prepared in the columella between the medial crura extends down to the nasal spine. F. Bone strut being placed in the columella. G. Diagram showing position of columellar strut supporting distal end of the dorsal bone graft. Note suture approximating medial crura. The technique illustrated in Fig. 19 may be combined with this procedure.

Fig. 19. Elongation of the columella.

A. and B. Flat nose due to short columella, a condition frequently observed in patients with bilateral cleft tip. C. Incisions outlining flaps. D. Mobilization of flaps to lengthen columella. E. and F. Closure of secondary defects by the V-Y method and medial displacement of the base of the alae.

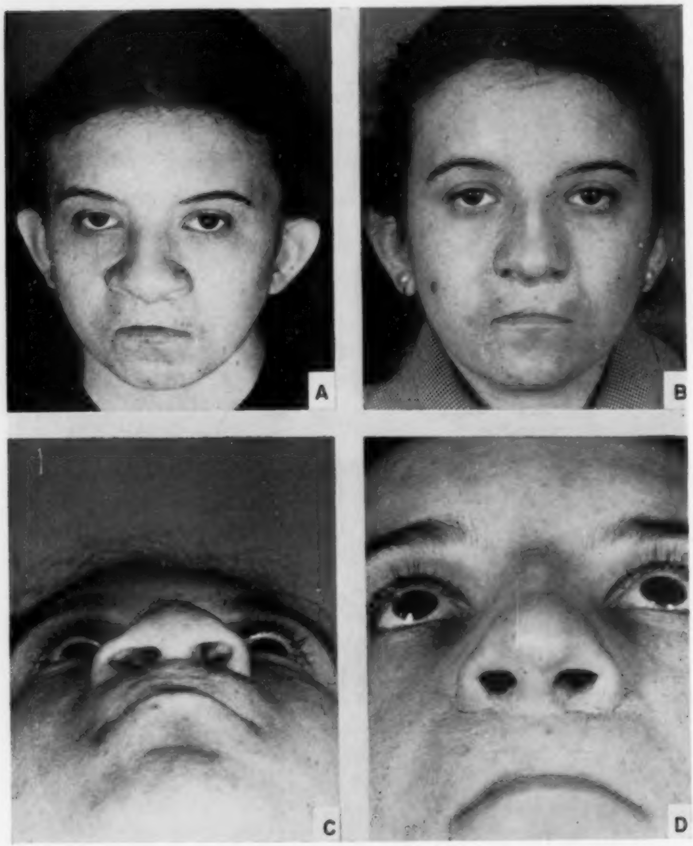


Fig. 20. The flat nasal tip.
A. and C. Example of flat nasal tip with short columella and wide nares in patient with bilateral cleft lip. B. and D. Result obtained by elongation of the columella using technique illustrated in Fig. 19 and increasing the projection of the tip by technique shown in Fig. 18.

Correction of the Excessively Pointed Tip.

A sharp narrow-tipped nose presents an undesirable facial appearance. This deformity is due to the particular anatomical configuration of the domes. The dome ordinarily

forms a gently curved arch at the point where the lateral and medial crura meet; a pointed tip results if the crura meet at a sharp angle. The technique suggested by Aufricht (1940) may be used to correct this type of deformity. The alar cartilages are exposed by the rim incision technique. The alar cartilage can be divided without incising through the vestibular lining at the highest point of the dome.; a suitable segment of cartilage can then be resected; if necessary, two or even three small strips of cartilage are removed (see Fig. 21); the alar cartilages are then

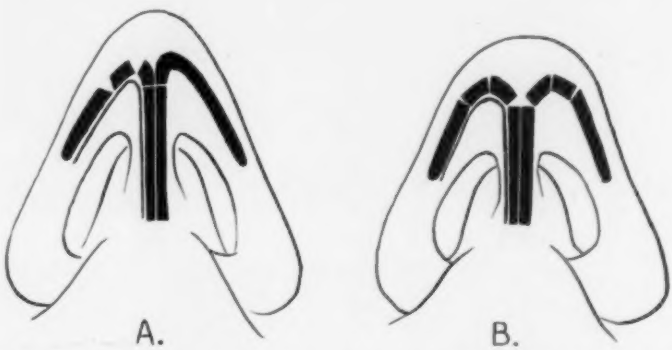


Fig. 21. Correction of pointed tip (after Aufricht). Excision of small segments of cartilage eliminates the sharp-pointed domes.

replaced against the overlying soft tissues. The mobile segments of alar cartilage bend downward and remain there to heal in position, assuming the normal arch curvature of the dome; careful support with intranasal gauze packing maintains the position.

Correction of the Prominent Tip.

An appreciable decrease in the projection of the tip is obtained by the procedure for correcting the pointed tip (see Fig. 21). The projecting tip may be adequate in shape or require only a slight modification, in addition to a diminution of its prominence. Strips of cartilage removed from the medial crura and from the lateral crura, lateral

to the dome, permit diminution of the projection of the tip (see Fig. 22). Careful diagnosis will occasionally reveal that the dorsum is not sufficiently projected and that the tip is in apparent protrusion. Fig. 23 illustrates a case in which the employment of a septal cartilage graft over the dorum remedied this condition.

Columellar Struts and Tip Implants.

If possible, implants should consist of autogenous cartilage; septal and alar cartilages are the grafts of choice.

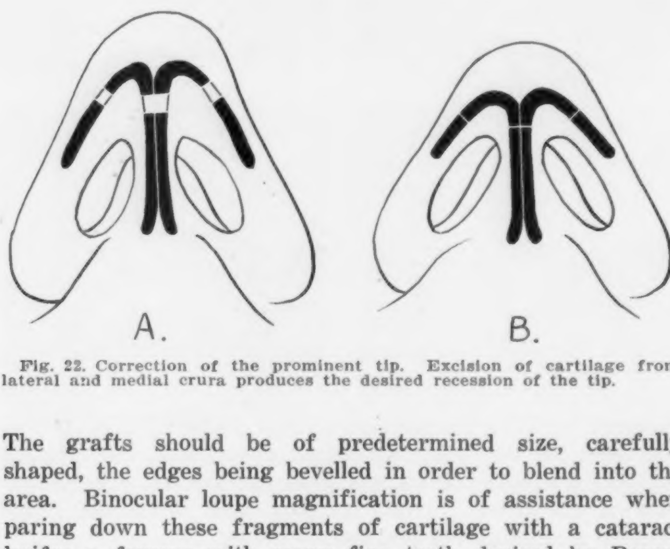


Fig. 22. Correction of the prominent tip. Excision of cartilage from lateral and medial crura produces the desired recession of the tip.

The grafts should be of predetermined size, carefully shaped, the edges being bevelled in order to blend into the area. Binocular loupe magnification is of assistance when paring down these fragments of cartilage with a cataract knife; a forceps with many fine teeth devised by Brown permits precise manipulation of the cartilage during the operation.

A septal cartilage implant at the base of the columella increases the protrusion of the columella or prevents retrusion when it is necessary to remove the lower portion of the septal cartilage (Converse 1950). The graft is placed through an incision along the anterior margin of

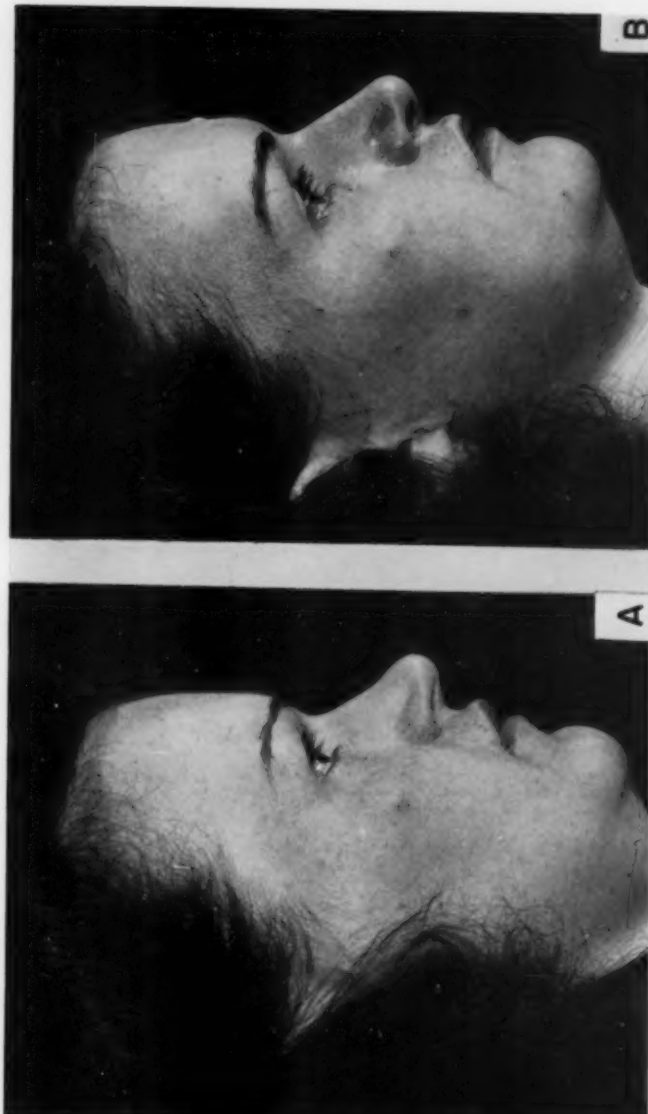


Fig. 23. Correction of prominent tip by increasing the projection of the dorsum of the nose. A. Preoperative view. B. Postoperative appearance after septal cartilage graft over the nasal dorsum.

the medial crus; this type of incision along the anterior margin of the medial crus is a routine procedure in our Clinic. The wound leaves no visible scar when carefully approximated with interrupted 5-0 plain catgut sutures.

In a round tip a slight increase in the projection of the lower portion of the tip may be obtained by a cartilaginous implant, trapezoid-shaped with its base above, placed in front of the area of junction of the medial crura and domes. A small flat piece of septal or alar cartilage implanted in this area creates a "facette" and reduces the round appearance of the tip. A small graft is also useful in this area in noses with bifidity if the bifidity cannot be completely corrected by approximating the medial crura.

We have not found that the placing of "buttons" of cartilage immediately above the area of junction of the domes gives a satisfactory result; these "buttons" tend to become displaced and to produce irregularity after postoperative edema has disappeared. Increase in projection of the tip is best achieved by direct action upon the alar cartilages, adequate shortening of the nose by excision of a triangular piece of cartilage from the lower portion of the septal cartilage, and adequate modification of the dorsum of the nose to a profile line which harmonizes with the tip. In noses with a markedly convex profile line, it is necessary to modify the profile line by diminishing its projection and reducing the line in such a way that the dorsum is lowered to a level of a line passing through the area of junction of the two medial crura.

The Bifid Tip.

Approximation of the medial crura and the domes usually serves to correct the bifid tip deformity (see Fig. 24). Exposure of the alar cartilages through rim incisions, removal of tissue between the medial crura, suitable incisions through the alar cartilages, excision of cartilage from the domes and suturing of the medial crura are the steps required for this type of corrective procedure.

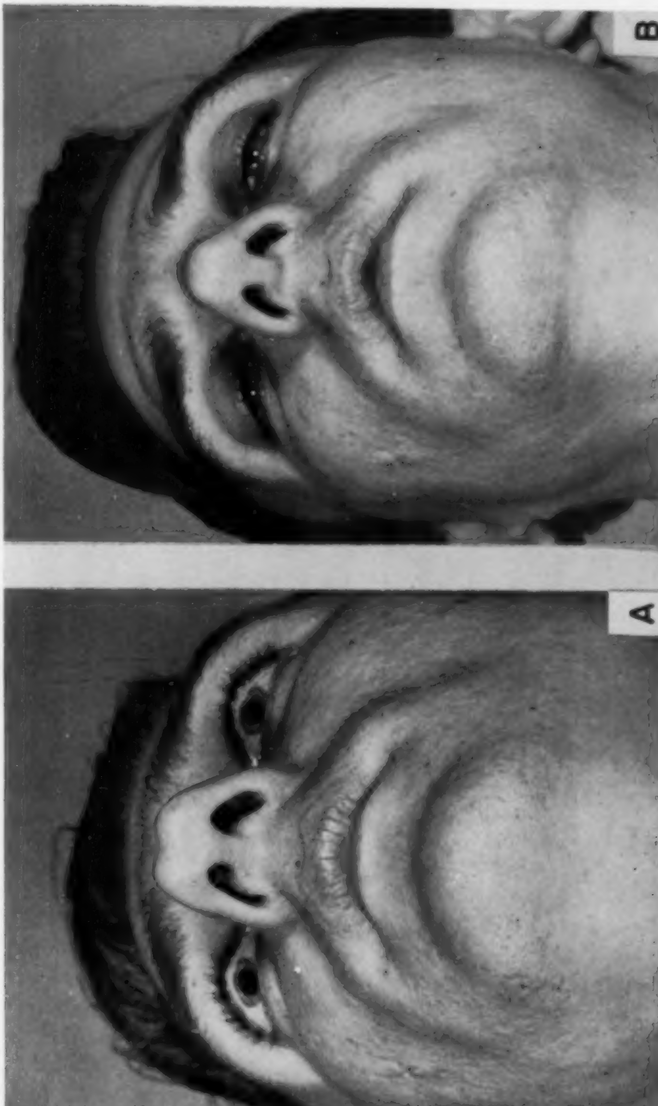


Fig. 24. The bifid tip.

A. Example of bifid tip characterized by separation between the medial crura. B. Result obtained by exposure of the alar cartilages by rim incisions and approximation of the medial crura.

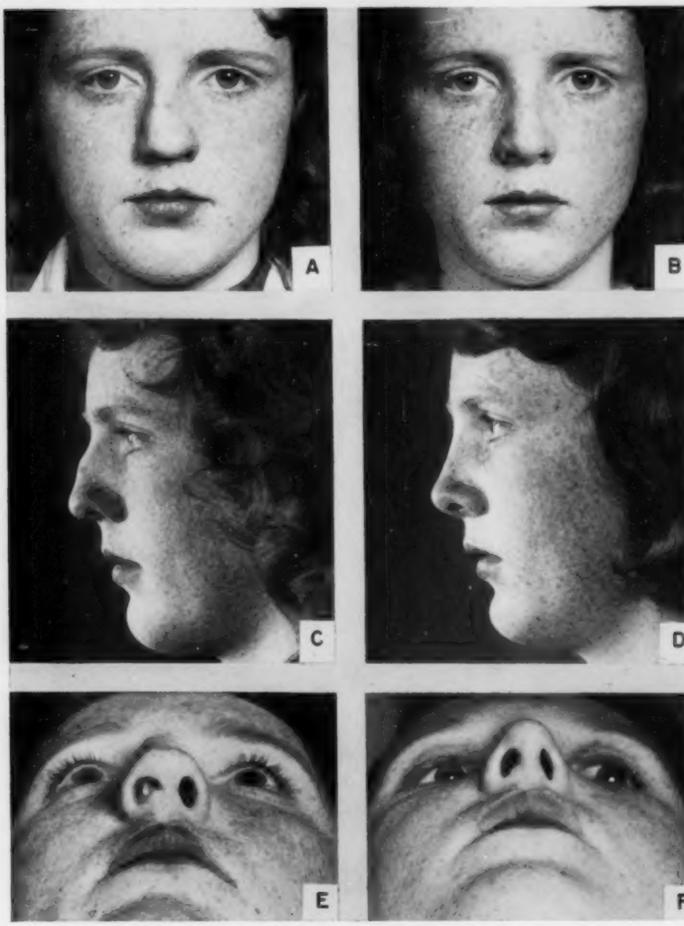


Fig. 25. The nasal tip in the deviated nose.

A. Widening of the tip by separation of the alar cartilages caused by deviation of the septal angle. B. Postoperative appearance. C. Hump deformity and retraction of the columella due to absence of septal support. D. Postoperative appearance after modification of the profile line and shortening of the nose. E. Characteristic deformity of the tip with widening. Note deviation of the septum and distortion of the columella. F. Postoperative appearance of the nasal tip.

The Deviated Nasal Tip.

The tip of the nose rarely shows marked deviation even in the most deviated nose. In some congenital malformations the area of junction of the two medial crura is nearly always in the midline, although some distortion of the lateral crura may be present due to deviation of the septal angle. Pressure of the laterally twisted septal angle may cause separation of the alar cartilages with bifidity of the tip and asymmetry, one lateral crus or dome being higher than the other (see Fig. 25-A, C and E). The pressure of the septal cartilage to one side of the columella results in lateral displacement of one medial crus with separation of the crura, widening and distortion of the columella, and lateral protrusion of the lower end of one of the medial crura (see Fig. 25-E).

Straightening the cartilaginous septum is an essential procedure to correct the nasal tip in such cases. It may be accomplished by a conservative technique; in other cases resection of the lower portion of the septal cartilage is necessary; a transplant of septal cartilage is placed into the columella to prevent retraction (Converse 1950).

Asymmetry of the lateral crura requires removal of a greater quantity of cartilage from one side than from the other; bifidity requires approximation of the medial crura (see Fig. 25-B, D and F).

A New Technique for Correction of the Wide Columella.

This condition is usually due to deviation of the lower edge of the septal cartilage and to lateral traction upon one medial crus, causing one medial crus to become separated from the other, and leaving a wide gap between the cartilaginous structures (see Fig. 26-A). Mattress sutures through the skin tend to cause an inflammatory reaction. To prevent this complication, the following technique has been used successfully—an incision is made along the anterior margin of the medial crus on each side in its lower portion (see Fig. 26-B); the overlying skin of each medial crus is then separated from the cartilage (see Fig. 26-C), and the

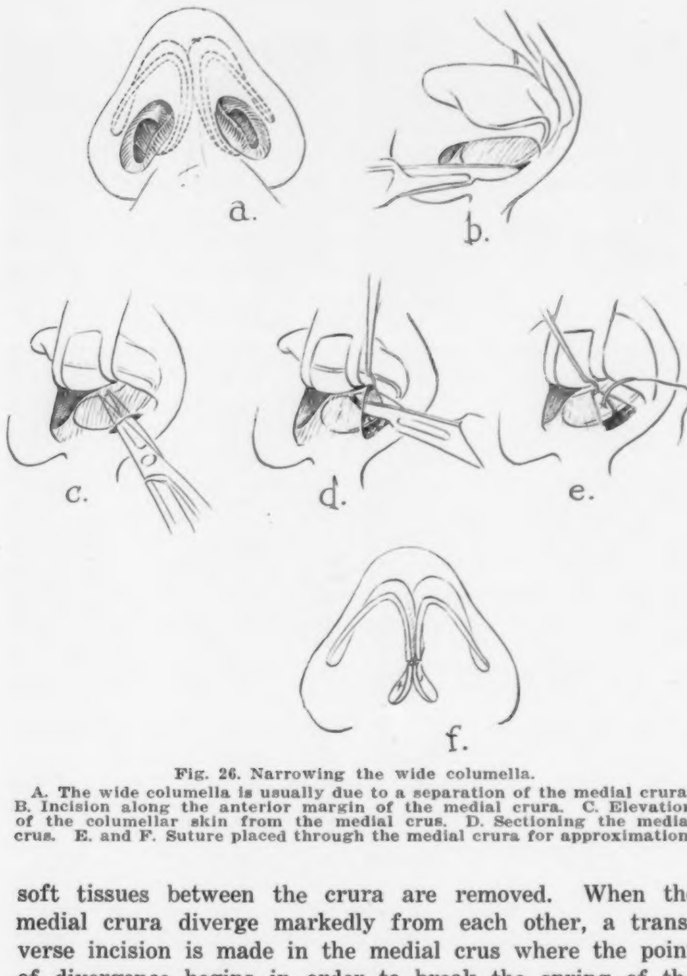


Fig. 26. Narrowing the wide columella.

A. The wide columella is usually due to a separation of the medial crura. B. Incision along the anterior margin of the medial crura. C. Elevation of the columellar skin from the medial crus. D. Sectioning the medial crus. E. and F. Suture placed through the medial crura for approximation.

soft tissues between the crura are removed. When the medial crura diverge markedly from each other, a transverse incision is made in the medial crus where the point of divergence begins in order to break the spring of the cartilage (see Fig. 26-D). A mattress suture of 5-0 chromic catgut approximates the medial crura (see Fig. 26-E and F); the suture is placed subcutaneously and in this manner does not ulcerate through the skin, the usual cause of inflammatory reaction.

COMPLICATIONS.

Complications are usually due to failure to achieve satisfactory shaping of the nasal tip by inappropriate, inadequate or excessive surgery. Some of these complications and their correction have been discussed previously in this paper (see Fig. 1 and 2).

Slight irregularities may be corrected by small cartilage grafts or by trimming protruding edges of cartilage. Secondary procedures should be instituted not earlier than four months after the primary operation to allow the edema to subside and the tissues to soften. The Safian or eversion techniques have provided the most satisfactory methods of approach for these secondary procedures. Inadequate contraction of the soft tissues and the pinched tip deformity are complications which require special consideration.

Inadequate Contraction of the Soft Tissues over the Modified Framework.

Excess skin and soft tissue over the modified nose framework is a frequent complication following corrective plastic surgery and occurs most frequently in the supra-tip area. Contraction of the soft tissues may extend for six months or as much as a year. Because the soft tissues fail to adapt themselves to the underlying profile line constituted by the newly formed dorsal border of the septal cartilage, a convexity of the supra-tip area results which negates an otherwise satisfactory result. The convexity in the supra-tip area is usually temporary in young individuals, due to soft tissue excess alone, for the skin is rich in elastic fibers and contraction occurs progressively; in older individuals, contraction may be inadequate. The age of the individual must be considered when planning reduction in the size of a very large nose; it should also be recalled that contraction of the soft tissues in older individuals may be incomplete.

Swelling in the supra-tip area may also be due to an excess of localized subcutaneous fat and muscular tissue, requiring subsequent removal. The eversion technique gives adequate exposure for the procedure. An incision is made

to expose the area. Careful removal of the subcutaneous tissue is accomplished with scissors, care being taken to avoid button-holing the skin or even to dissect the subcutaneous tissue at a too superficial level in order to avoid skin necrosis.

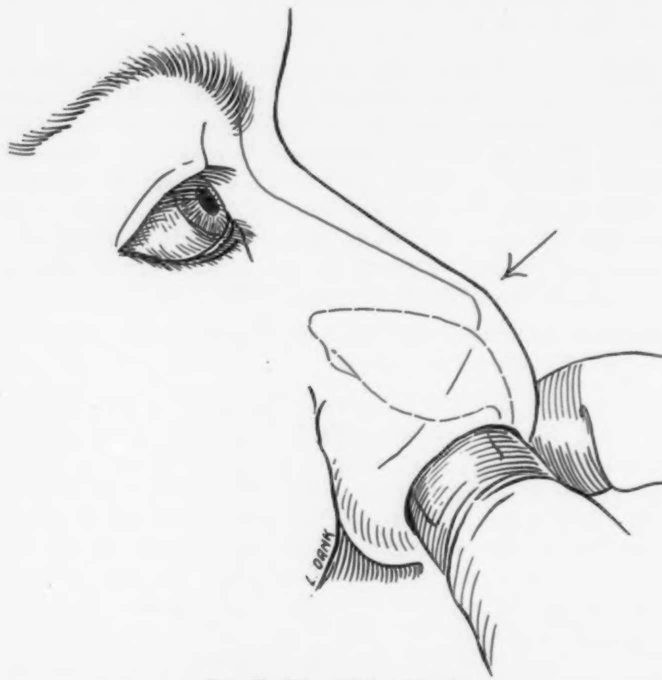


Fig. 27. The septal angle test.
A useful test to assist the surgeon in evaluating the amount of cartilage to resect from the septal angle. Traction downward upon the columella causes the septal angle to protrude under the skin of the supra-tip area if cartilage remains in excess in this area.

There may be considerable thickening and stiffening over the tip due to the development of sebaceous and sweat glands in the region. Seborrheic skin does not contract, and is unfavorable for satisfactory modification of the tip.

Convexity in the supra-tip area is often due to inadequate trimming of cartilage of the septal angle (see Fig. 27); it

may also be due to excessive removal of septal cartilage, forming a pocket between the septal angle and the alar cartilage (see Fig. 2).

Treatment of the Pinched Tip.

Excessive removal of cartilage from the domes results in a tip which is too pointed, a difficult condition to correct. When excessive cartilage has been removed from the lateral crus of the alar and lateral cartilages, a pinched deformity with stenosis of the nasal airways results, particularly if an excess of vestibular lining has also been removed (see Fig. 28-A, C and E).

The pinched nose with vestibular stenosis presents a dual problem: *1.* deficiency of the lining, which causes an inward pull upon the external nasal structures must be remedied, and *2.* absence of cartilage which requires support. Such cases have been successfully treated by the following procedure: Intranasal scar tissue is removed, and cicatricial bands are eliminated by a Z-plasty procedure. The remaining intranasal raw area is then covered by means of full-thickness skin grafts from the upper eyelids. This skin is preferred because it is thin skin, devoid of hair follicles and is readily available. An alternate source of grafts is the skin situated on the posterior aspect of the auricle, also relatively thin skin. Dental compound is carefully softened in warm water, then applied over an alcohol lamp flame after being covered with a layer of paraffin ointment. The compound is rolled into a cigar-shaped mass which is carefully pressed into the nasal vestibule. The mold is then hardened in sterile ice water. A mold of compound is made for each vestibule and is duplicated. One of these serves to carry the skin graft which is placed over the mold, raw surface outward. The alternate mold is employed in the construction of a thin acrylic prosthesis. The dental compound mold is removed after a period of seven days and is replaced by the prepared acrylic prosthesis. These molds can be shell-like in thickness and are inconspicuous when worn inside the nose; they are designed to fit into the recess of the vestibule above and behind the internal narial

Fig. 28. The pinched tip due to excessive removal of alar cartilage and vestibular lining.

A. and B. Typical appearance of pinched tip following an operation by an unqualified practitioner. Note also the inadequate osteotomy and profile line correction, the retraction of the columella and drooping of the tip. C. and D. Correction obtained by secondary osteotomy and modification of the profile line, autogenous septal cartilage graft in the columella. The deficient nasal lining was restored and small thin acrylic molds employed for support. E. Typical pinched tip. F. Result obtained. Note intra-vestibular molds visible in photograph. These acrylic molds were designed and fitted by Dr. Harry H. Shapiro.

fold below. The prostheses should be worn for a long period of time because of the probability of eventual contraction of the intranasal skin graft; when the cartilage deficiency is extensive they should be worn permanently. Fig. 28 illustrates a case in which this type of treatment was employed to eradicate a deformity produced by excessive removal of cartilage and vestibular skin.

BIBLIOGRAPHY.

AUFRICHT, G.: A Few Hints and Surgical Details in Rhinoplasty. THE LARYNGOSCOPE, 53:317, 1943.

BECKER, O. J.: Principles of Otolaryngologic Plastic Surgery. Published by Amer. Acad. Ophthal. and Otol., 1952.

CONVERSE, J. M.: Technique of Bone Grafting for Contour Restoration of the Face. *Plastic and Reconst. Surg.*, 14:332, 1954. Corrective Surgery of Nasal Deviations. *Arch. of Otol.*, 52:671, 1950.

DEKLEINE, E. H.: Nasal Tip Reconstruction Through External Incisions. *Plastic and Reconst. Surg.*, 15:502, 1955.

ERICH, J.: A technic for Correcting a Flat Nostril in Cases of Repaired Harelip. *Plastic and Reconst. Surg.*, 12:320, 1953.

FOMON, S., and SYRACUSE, V. R.: Routine Technic of Typical Rhinoplasty. *Ill. Med. Jour.*, 94:242, 1948.

FRED, G. B.: The Nasal Tip in Rhinoplasty. *Ann. Otol. Rhinol.*, 59:215, 1950.

GOLDMAN, I. B.: New Technique for Corrective Surgery of the Nasal Tip. *Arch. Otol.*, 58:183, 1953.

JOSEPH, J.: Nasenplastik und Sonstige Gesichtplastik. Kabitzsh C., Leipzig, 1931.

NÉLATON, C., and OMBRÉDANNE, L.: La Rhinoplastie, Steinheil, G., Paris 1904.

RETHI, A.: Raccourcissement du nez trop long. *Rev. de Chir. Plast.*, 2:85, 1934.

SAFIAN, J.: Corrective Rhinoplastic Surgery. Hoeber, P. B., 1935.

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RECOVERY FROM ACOUSTIC TRAUMA IN THE GUINEA PIG.*†

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INTRODUCTION.

This paper describes one of a series of studies undertaken to explore more thoroughly the significant parameters of acute acoustic trauma and to evaluate some of the methods used to study acute acoustic trauma in the guinea pig.

In one of the earlier studies[1] in this series, ears were exposed to tones generated by a siren. Some of the specimens were obtained immediately after exposure, but most of the guinea pigs were shipped alive from the location of the siren at Wright-Patterson AFB, Ohio, to Washington University Medical School in St. Louis, Mo., before being sacrificed to obtain the temporal bone specimens. For this reason the pathology during the first week after exposure was largely neglected. In another series[2] all of the exposures were made with electrodes in place and the specimens taken immediately.

The present series of experiments was designed to relate the pathology, observed during the first week after trauma, both to the pathology in acute experiments and to the pathology at longer intervals after trauma. Both electrophysiological and histological methods are used to evaluate the injuries. The primary method is histological and the

*Supported by Contract No. AF 18(600)-58 between Washington University and the Bio-Acoustic Branch of the Aero Medical Laboratory, Wright Air Development Center.

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Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication October 29, 1956.

electrophysiological results are compared to this standard. In order to evaluate the possible influence of the surgical techniques required by the electrophysiological method on the observed pathology, parallel experiments omitting this surgery are included.

In these new studies it is assumed that any injury to the organ of Corti is significant but not necessarily proportional to loss of hearing function. Other experiments[3,4] employing conditioned response techniques indicate that hearing function is not related to degree of observed injury in any simple way.

PHYSIOLOGICAL AND ANATOMICAL METHODS.

Young adult guinea pigs (200-300 g) were anesthetized with Dial (Ciba) in urethane (0.50 cc per kilo, intraperitoneally). The ears, which were to be examined by both electrophysiological and histological methods immediately after acoustic trauma, were opened by a ventrolateral approach[5] that exposed the bulla. The bulla was opened and nichrome steel electrodes were inserted in scala tympani and scala vestibuli of the basal turn, in scala vestibuli of the third turn and in the apex,[6] and fixed in place with dental cement on the edge of the opened bulla. A metal ear-speculum was securely sewn into the ear canal. The open end of the speculum was firmly seated in a length of garden hose connected, as a closed system, to an Atlas PM-25 loudspeaker driver unit. The electrodes were connected through cathode follower circuits and a balancing network[6] to allow simultaneous differential recording of the cochlear microphonics (CM) from the basal and third turns and of the nerve action potential (AP) from the cochlea as a whole. Thresholds and the maximum potentials produced by non-injurious sound pressure levels (approximately 110 db*) were determined; *a.* for the overall AP produced by 500 cps and 8000 cps tone pips; *b.* for the CM produced in the third turn by a 500 cps tone, and *c.* for the CM produced in the basal turn by a 2000 cps tone. These CM measures have been pre-

*Our acoustic reference level is 0.0002 microbar throughout.

viously shown to be related to the condition of the organ of Corti[2]. The AP measures were made for other reasons and will be reported separately. The ears were then exposed for one minute to a 170-cps or a 545-cps tone at sound pressure levels ranging from 136 to 156 db. The same thresholds and maximum voltages were again measured immediately after exposure and verified one hour after exposure. These exposures were expected to produce permanent injuries in the third turn at or near one pair of electrodes and lesser or no injury in the basal turn at the site of the other pair of electrodes.

Those ears that were to be examined on another day and those ears to be examined only by the conventional histological method were similarly exposed with the speculum sewn or held securely in place, and the animals were allowed to recover from the anesthesia. After one, two, seven or 14 days those animals that were to be examined by both methods were again anesthetized, electrodes were placed and the post-traumatic thresholds and maximum voltages determined as described above.

Forty-two ears were examined by both methods, and 33 additional ears were examined only histologically without the complications of surgery and insertion of electrodes. The animal under anesthesia was bled and the circulatory system washed with physiological saline solution. Fixing solution (Heidenhain-Susa) was immediately injected and the temporal bone removed for fixation, decalcification and subsequent sectioning. Every fifth section was mounted for microscopic examination. The methods of perfusion, fixation, sectioning, etc., were the same as those used in earlier phases of this investigation.[1,7] The microscopic examinations were generally confined to mid-modiolar sections of the cochleas except to assess possible local damage done by the drills or the electrodes.

ACOUSTIC EXPOSURES.

Acoustic trauma was produced by the same electro-acoustic system employed for measuring the electrical responses. We

had intended to confine the sound pressure levels of the exposures to 142 to 146 db, the threshold described earlier[2] for permanent injury from a one minute exposure. After about three-quarters of the exposures had been completed a check calibration of the electro-acoustic system revealed that the previously published calibration[2] was only approximately correct as used in these experiments. Sound pressure levels were measured, as before, just in front of the tympanic membrane by means of a probe-tube microphone made from a 20-gauge spinal puncture needle[8]. It was found that minor changes in the coupling of the system could produce changes in sound pressure level that ranged from six db below to ten db above the previous average calibration. Since we could not control these differences reliably, we can only say that the exposure sound pressure levels lay between 136 db and 156 db. Some evidence adduced from the normal thresholds indicates the levels were usually higher rather than lower than the intended values of 142 to 146 db.

RESULTS.

Normal Values.

These experiments raised the question of normal values for the electrophysiological potentials. In previous experiments pre-exposure thresholds for CM and maximum potentials for CM were routinely measured. So long as these values were reasonable and so long as the configurations of the responses were qualitatively normal, the ears were accepted for experiment. The losses for the CM potentials were determined directly by subtracting post-exposure thresholds and maximum potentials from the pre-exposure values. For the present series, in order to be able to evaluate injury from post-exposure potentials alone, it was necessary to establish normal pre-exposure values.

Although threshold values for CM are subject to the uncertainty of calibration described above, the maximum CM potential obviously depends little if any on this calibration. It was possible to compare the maximum CM potential ob-

served for the small group (N = 8) of pre-exposure ears in this series to those observed in a subsequent series of 52 normal ears measured using a more precisely calibrated acoustic system. These comparisons for the maximum CM potential produced by a 500 cps tone in the third turn and for a 2000 cps tone in the first turn are shown in Table I, together with the interquartile range for the larger series.

TABLE I.

Normal Maximum Cochlear Microphonic Potentials.
(Voltage levels in decibels above 1 microvolt peak to peak)

	Pre-exposure Series N = 8 Median	Later Series N = 52 Q1	Later Series N = 52 Median.	Later Series N = 52 Q3
500 cps tone	55 db	53 db	55 db	58 db
2000 cps tone	70 db	67 db	69 db	71 db

It is clear that the smaller series contained in the present experiments is a good sample of the larger more recent series. Accordingly the median values of the maximum CM potential have been used as the normal values to which all post-exposure values were compared. We cannot compare the median values for the CM threshold in the same way but have assumed that the small sample is also adequate in this dimension. Our values for threshold shift were obtained by subtracting post-exposure thresholds from the median pre-exposure threshold observed for the sample of eight ears.

CORRELATION OF ELECTRICAL INDICES WITH LOCAL PATHOLOGICAL CHANGES.

Previously in acute experiments[2] we have established general relationships between the degree of local injury and the decibel losses, both in sensitivity and in high-level output of the cochlear microphonic. Differential recording from paired intracochlear electrodes allows measurement of the cochlear microphonic produced by a small segment, three to four millimeters, of the basilar membrane. For this reason the loss of electrical potential is specific for the in-

jury to the portion of the organ of Corti between the electrodes.

The same nine-point scale[2] for rating the severity of the anatomical injury to the organ of Corti has again been used. Briefly, 1 and 1 + indicate "normal" and "within normal limits"; 2 and 2 + indicate minor degrees of exudate, vacuolization of cytoplasm in hair cells or mild changes in the nuclei of hair cells; 3 and 3 + indicate larger vacuoles, swelling and disintegration of cells and nuclei, and extensive

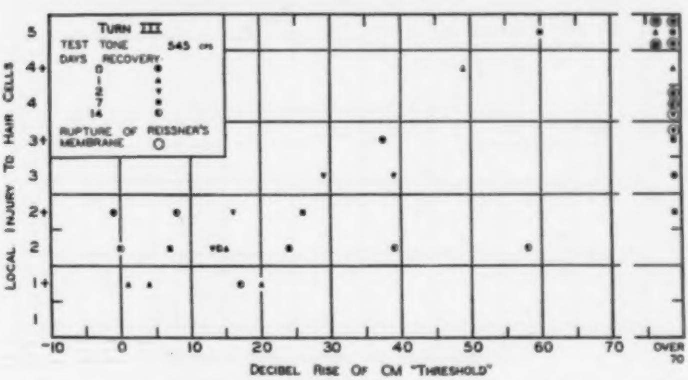

Fig. 1. Local injury to the hair cells in the third turn plotted against decibels rise of CM "threshold" for 500 cps in the third turn. The symbols correspond to the different durations of the recovery period. For injury scale see text.

loss of mesothelial cells; 4 and 4 + indicate partial absence of hair cells or their nuclei and disruption of supporting structures, often including rupture of Reissner's membrane; 5 indicates absent hair cells, separation of the organ of Corti from the basilar membrane, and often complete absence of the organ of Corti.

The relation of the condition of Reissner's membrane to the observed potentials is obscure. It is not always easy to assign to a particular injury a value on the injury scale when Reissner's membrane is ruptured. Occasionally relatively normal-appearing hair cells will be accompanied by rupture

of Reissner's membrane. The practical implications of such a combination are not clear. Arbitrarily we have rated the injuries primarily on the condition of the cells of the organ of Corti and then noted separately when Reissner's membrane was injured. The results are contradictory: in some instances where rupture occurred with relatively slight changes in the organ of Corti, there was total loss of electrical potential; in other instances, the losses with rupture were small. We have no satisfactory explanation for this.

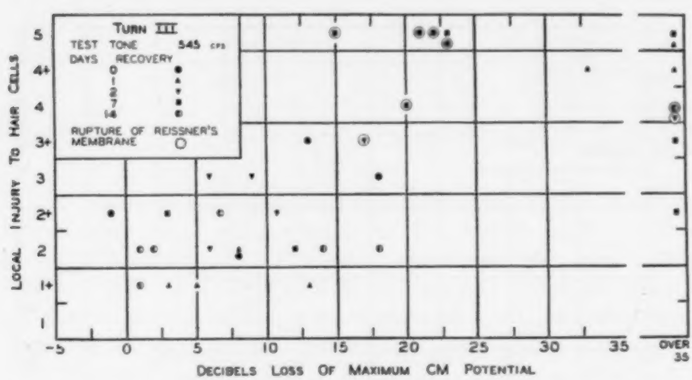

Fig. 2. Local injury to the hair cells in the third turn plotted against decibels loss of maximum CM output for 500 cps from the third turn.

The degree of injury observed anatomically in the *third* turn is plotted against the decibel rise in CM threshold in Fig. 1, and against the decibel loss of maximum CM potential in Fig. 2. All durations of the recovery period are presented together but are identified as separate parameters by the different symbols. These relationships compare favorably with those previously reported[2] for acute injuries. The duration of the recovery period does not appear as an important parameter.

A new finding is the relation between the degree of injury observed anatomically in the *first* turn and the electrophysiological potentials. Degree of injury is compared to

decibels rise in CM threshold in Fig. 3, and to decibels loss of maximum CM potential in Fig. 4. The organ of Corti in the first turn of all ears was "normal" (1) or "within normal limits (1 +) when examined microscopically unless there was also rupture of Reissner's membrane. Six of these essentially normal ears showed threshold losses of 21, 23, 29, 30, 32, and 33 db, and respective losses in maximum potential of 4, 11, 12, 10, 11, and 12 db. On the basis of these losses we expected to find discrete minor injuries.

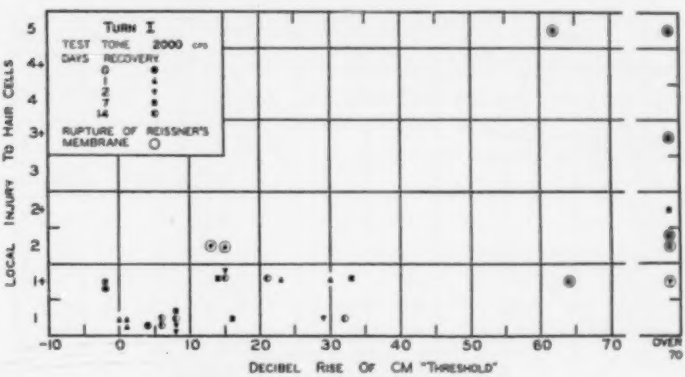

Fig. 3. Local injury to the hair cells in the basal turn plotted against decibels rise of CM "threshold" for 2000 cps in the basal turn.

Previously[2], threshold losses of 20-30 db and losses of maximum potential of 10-15 db were associated with injury ratings of "2". We believe the absence of these minor injuries in the present series may be significant and have considered several possible explanations for this finding.

The possibility that anatomic recovery may significantly precede physiological recovery is an attractive hypothesis that might explain the discrepancy. An ear judged "within normal limits" one or two weeks after trauma might earlier have shown evidence of moderate injury, whereas an ear judged "within normal limits" on the same day or one or two days after trauma might never have shown any

evidence of injury. It is reasonable to assume that these ears which appear the same might function differently and show different losses in their electrophysiological potentials. Statistical analysis, however, failed to reveal a relation between duration of the recovery period and these losses.

A second hypothesis assumes that even though the first turn of the cochlea appears essentially normal (1 or 1+) the electrical potentials produced in the first turn may be influenced by the severity of the injury in the third turn;

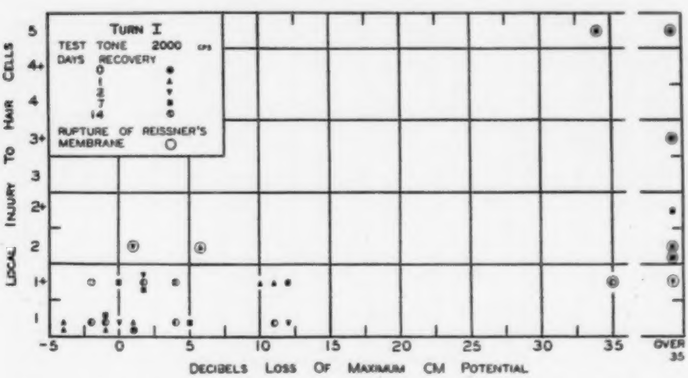

Fig. 4. Local injury to the hair cells in the basal turn plotted against decibels loss of maximum CM output for 2000 cps from the basal turn.

however, the threshold losses found when the first turn was essentially normal did not correlate significantly (r=.18, 5%=.45) with the threshold losses measured in the third turn or with the degree of injury found anatomically in the third turn (r=.05). This lack of correlation also argues against the existence of any other variables, such as anesthesia or hypoxia, which should produce similar changes in the physiology of both turns.

Another hypothesis, not subject to test in these experiments, depends upon the fact that, during stimulation by a low frequency tone, adjacent segments of the basilar membrane in the first turn move in phase while those in the

third turn move slightly out of phase. Possibly the kind of anatomical injury that can be related to losses in electrophysiological potentials depends to some extent on the presence of the shearing forces that must accompany longitudinal bending of the basilar membrane.

A fourth hypothesis, which is also not subject to test here, retreats to the assumption of "sub-microscopic" injury. This hypothesis would include biochemical depletion of the hair cells or possibly minor displacements of the hair cells which we cannot distinguish from sectioning artifacts.

PATHOLOGICAL CHANGES.

This series of experiments was designed to have features comparable to each of two previous series[1,2] and also to provide some internal control on the possible influence of the insertion of electrodes on the pathology observed after acoustic trauma.

The methods used for the present series are nearly identical to those previously reported by Davis et al.[2] in regard to the acoustic system, the exposure frequencies, the exposure intensities, the exposure duration and the electrophysiological measurements. The methods differed to the extent that the majority of the exposures in the present series were made with the bulla intact and the ear was allowed to heal for varying intervals after the acoustic trauma. In the method employed by Davis et al., the exposures were made with the bulla open and the electrodes already in place. The temporal bone specimens were taken approximately one hour after the acoustic trauma.

The histological changes found in the cochlea were quite similar. The extent, the location and the severity of the lesions were the same so long as the injuries were not complicated by rupture of Reissner's membrane. In the present series rupture was occasionally accompanied by substantial injury to the whole basilar membrane from round window to apex. The more severe injuries were still centered in the third and fourth turns. These constitute the most extensive injuries encountered to date. In the previous series

the incidence of rupture of Reissner's membrane was slightly higher, and when it occurred was usually associated with an increase in apical injury. A loss of mesothelial cells from the tympanic surface of the basilar membrane, particularly in the third and fourth turns, was similar for each series. Small amounts of red blood cells, usually in scala vestibuli were found with about the same incidence.

The principal differences noted were in the states of degeneration and repair observed in the cochlea. These secondary changes, of course, did not appear in the previous series.[2] Proliferation of low cuboidal epithelium over a denuded basilar membrane occurred at the earliest between one and two days following the exposure and appeared to be complete within one week. Regeneration of mesothelial cells occurred more slowly or occasionally not at all. Degenerative changes in peripheral nerve fibers and spiral ganglion cells usually appeared two weeks after exposure.

The methods used for the present series were like those used by Covell and Eldredge[1,7] in that the bulla was intact during exposure, and the ears were allowed to recover for varying periods following exposure. The methods were different in that Covell and Eldredge used a siren sound source, higher frequencies, often higher intensities, and no electrophysiological measures on the ears exposed to the siren; furthermore, few ears were examined less than one week following acoustic trauma, while many were examined after intervals of more than two weeks.

A number of systematic differences appeared between the injuries for these two series. These differences are related to the portions of the basilar membrane most strongly stimulated by the different exposure frequencies used. The chief difference, as would be expected, between the siren exposures, ranging from 4 kcps to 40 kcps, and the present series was the location of the injury along the basilar membrane. In the siren experiments the lesions were found to occur in the lower one-half of the cochlea with only minor lesions, if any, in the third turn of the cochlea. As the frequency of the exposure tone was increased the lesions

moved further toward the round window. In the present series the upper one-half of the cochlea was involved, and frequently lesions were discernible along the entire extent of the basilar membrane with a tendency for the more marked injuries to involve the fourth turn of the cochlea.

Other differences which may have importance were observed. In the present series, the internal hair and supporting cells of the first turn frequently revealed nuclear and/or cytoplasmic alterations in the absence of other lesions at this location. Degeneration of the connective tissue cells of the limbus was common in the turns apical to the maximum injury, the third and fourth turns, after siren exposures but was rarely seen at any location in the present series. In the present series rupture of Reissner's membrane was more frequently observed, as was also thinning and even a separation of the two layers of this membrane. The mesothelial cells on the tympanic surface of the basilar membrane were consistently more affected. For the present we believe these differencs also are related to the different patterns of vibration of the basilar membrane.

The other differences that were observed are logically related to the different lengths of time allowed for the ears to recover after exposure. As noted above, the earliest that low cuboidal epithelium replaces missing organ of Corti is about one or two days after exposure. Other clues to the recovery sequence are somewhat less objective.

It is necessary here to distinguish between processes that can be considered recovery and those that are healing in the sense that "scar" of inactive tissue has covered over a gap of injured cells. The evidences seen for healing are relatively straightforward. The low cuboidal epithelium which covers a denuded basilar membrane is a good example. A somewhat thinned layer of mesothelial-like cells similarly appears on the tympanic surface of the basilar membrane. Club-like formations may be found on the torn edges of the ruptured Reissner's membrane.

Recovery of mildly injured cells to their normal state

both anatomically and physiologically is more difficult to determine. Limitations inherent in the methods of preparing the specimens and in the resolution of light microscopy often leave unanswered questions about the probable condition of specific cells. Several observations, however, lead us to believe that true recovery often occurs. The acutely injured but intact organ of Corti may reveal minor changes in hair cells and a swelling and vacuolization of supporting cells. The internal phalangeal cells are particularly susceptible to such changes. Corresponding cells examined a few days after trauma show a more normal appearance. Immediately after trauma there often appears to be an injury to the distal ends of Deiters' cells in the area of Deiters' cup. Alexander and Githler[9] have previously described dislocations between hair and supporting cells in this location. This injury is followed on the first and second days after trauma by swelling and enlargement of Deiters' cells to such an extent as to impart an appearance of shorter external hair cells. Protoplasmic extensions of supporting cells may bulge between the external tunnel rods into the tunnel of Corti. A week after trauma these extensions can become detached from the cell and spheres and globules are left in the tunnel. The associated injured cell has by this time either returned to a more normal appearance or further degenerated. In some specimens the degree of injury to the organ of Corti was less than usually found with loss of mesothelial cells directly beneath it. As noted above mesothelial cells are replaced rather slowly. It is possible that these cases represent recovery of minor injury to the hair cells that precedes replacement of the mesothelial cells.

The appearance of the overall lesion along the basilar membrane one week or more after trauma also provides circumstantial evidence that the so-called minor anatomical changes may either disappear or else go on to further degeneration. Injuries examined immediately after trauma show a gradual gradation of normal to maximally injured organ of Corti along the length of the basilar membrane. Later the injuries tend to show a much narrower range of gradation. The scale by which the injuries can be rated

tends to be reduced to "normal", "moderately degenerated" and "severely degenerated" or "absent"; further, the boundaries of areas so classified are rather sharp. We assume these "boundaries" are emphasized by the recovery of cells on one side and further degeneration of cells on the other.

Within the present series the exposures for those ears which were subsequently opened for implantation of electrodes are directly comparable to the exposures received by the unoperated ears. The injuries showed about the same range of variation for each kind of experiment. The extent of the lesions along the basilar membrane, their nature and severity were about the same for the two groups. The only noticeable difference was the presence of a few red blood cells, usually in the scala vestibuli in or near the turns into which electrodes had been inserted. There were no red blood cells in the scalae of the cochleas of the nonoperated animals. The incidence of rupture of Reissner's membrane was approximately the same for operated and nonoperated ears, and so was loss of mesothelial cells. There was no apparent difference in the incidence of apical injury between the two groups. Approximately one-third of all specimens revealed rupture of Reissner's membrane. When rupture was found to have occurred the severity of the apical lesion was usually markedly increased.

DISCUSSION.

The relative importance of specialized anatomical structures of the cochlear duct to the maintenance of normal function of the organ of Corti is not entirely clear. Reissner's membrane is a biochemical[10] and electrical[11,12] barrier. Rupture of the membrane should allow mixing of endolymph and perilymph and shunting of at least one physiological potential. We would like to assume that this barrier is critically important to normal electrophysiological function of the ear. As noted earlier, the losses of potentials associated with rupture of Reissner's membrane were inconsistent and do not improve our understanding of normal function of the ear. Mixing may even occur without a complete rupture. An apparently intact membrane may be thinned due

to stretching, the two layers of the membrane may be split apart or minute ruptures may be present and the permeability of the membrane correspondingly altered. In our studies we have come to recognize thinning, splitting, etc., as effects of trauma even though they may also occur as artifacts. The layer of elongated cells that is continuous with the lining of the scala vestibuli is frequently torn while the underlying epithelial layer of the cochlear duct remains intact. The nuclei and cytoplasm of cells of both layers show the effects of an abnormal amount of bowing or stretching of the membrane. The nuclei may be pycnotic and cell boundaries indistinct or they may stain feebly in comparison with other areas of the membrane not subjected to the same stress.

Degeneration of connective tissue cells in the limbus is one of the puzzling changes associated with the pathology of acoustic trauma. The location of this finding is usually not at the same level as the most severe injury to the organ of Corti. It is most frequently found after exposures to frequencies above 1000 cps. A similar degenerative change is sometimes found in the tympanic part of the spiral ligament. The latter lesion, however, is usually in the turn of maximum injury and does not seem to be related to the frequency of the tone used to produce trauma. The degenerative change in the limbus has been noticed by Schuknecht[13] in the cochleas of senile cats, but it is an uncommon finding in sections of cochleas of senile guinea pigs.[14] The illustrations accompanying the article by Riskaer et al.[15] on the effects of neomycin on the guinea pig cochlea show the same changes in the limbus. It is doubtful that this finding has much bearing upon the function of the organ of Corti unless it can ultimately result in shrinkage of the limbus with consequent dislocation of the anchorage for the tectorial membrane. The epithelial border of the limbus and Huschke's teeth, upon which the thin portion of the tectorial membrane rests, are usually found to be intact even when there has been a degenerative change in the connective tissue cells beneath them. We believe[16] that in movement of the cochlear partition by intense sound stimulation, the point behind the attachment of Reissner's membrane to the

limbus is a vulnerable spot, and the endothelial-like cells of the lining of scala vestibuli covering the limbus at this point may rupture. Perilymph from the scala vestibuli may then enter the substance of the limbus. It is possibly significant that rupture of Reissner's membrane does not appear in the same turns as degeneration of these connective tissue cells. The membrane, however, may be bowed and thinned indicating the presence of stress. The combination of these observations and Békésy's description[17] of relative motions among the components of the basilar membrane suggest that rupture of Reissner's membrane may be associated with predominantly perpendicular displacements of the organ of Corti, whereas injury to the attachments of Reissner's membrane may be associated with predominantly longitudinal, or occasionally radial, displacements of the organ of Corti.

The significance that can be attached to loss of mesothelial cells on the tympanic surface of the basilar membrane is questionable. This lesion is most often seen in the same areas as the more severe injuries to the organ of Corti. The mesothelial cells appear to have been stripped from the membrane and rolled into a cylinder. Proliferation of cells which are continuous with the lining of scala tympani over such a denuded area frequently occurs in about a week following the exposure. Since we now believe that the tunnel of Corti and other spaces of the organ of Corti contain perilymph, we also believe that the mesothelial cells have no functional importance as a part of semipermeable membrane. In support of this view we have observed normal organ of Corti remaining in areas where the mesothelial cells were torn away. Similarly sections through cochleas of senile guinea pigs[14] often show a loss of mesothelial cells in the second to fourth turns inclusive. This loss is presumably the result of degenerative changes with age and is not always accompanied by marked change in the sensory or supporting cells of the organ of Corti.

The reticular lamina is usually regarded as being particularly resistant to fracture by force. It was found in dissection of pieces of organ of Corti in the fresh state[18]

that hair and supporting cells remained attached to the reticular lamina while other attachments were lost. We have found that the internal hair cell may occasionally lose its insertion in the reticular lamina and burst out of the organ of Corti following exposure to intense sound. We regard the reticular lamina as an important part of the barrier between the endolymph of scala media and the perilymph within the organ of Corti and thus of great functional importance physiologically as well as mechanically. Mildly injured cells which normally would be expected to recover may well degenerate in the presence of any injury to the reticular lamina which reduces its effectiveness as a barrier. We have observed small tears in the reticular lamina after traumatic exposures. Some of these are undoubtedly artifacts. Others are accompanied by degenerative changes in the sensory cells that are out of proportion to the concomitant minor injuries to the supporting cells. We believe these tears are real and can account for the observed pathology.

It is unfortunate that because of technical difficulties in the preparation of sections of cochleas there is no way in which to judge a change in position and shape of the tectorial membrane. Accompanying severe lesions to the organ of Corti the tectorial membrane has sometimes been torn from its limbic attachment. There is little doubt that dislocations of it in relation to hair and supporting cells do occur and it is not possible to predict the effects of such malposition upon function. It was shown, in dissection of the cochlear duct in fresh specimens, that the tectorial membrane can be readily lifted from the organ of Corti and that its connections with the hairs of the sensory cells can easily be broken.

A fracture or extreme bowing of the tunnel rods has in certain instances been considered to be adequate to injure neighboring cells. Schnukecht[19] believes that the external hair cell closest to the external tunnel rod is most frequently injured in acoustic trauma. Less readily detectable is any injury to the fine nerve fibers that pass between the tunnel rods.

We believe that there exist some cellular changes of importance which are not readily visible with light microscopy. Examples of these are minor injuries, *1.* to nerve endings and base of external hair cells in the region of Deiters' cup; *2.* to the reticular lamina; *3.* to the tectorial membrane; and *4.* small cellular dislocations that are difficult to distinguish from the artifacts due to sectioning, embedding and fixation. It is possible that some of the electrophysiological losses in the basal turn associated with normal organs of Corti can be explained on this basis.

SUMMARY.

Guinea-pig ears have been examined anatomically and for changes in electrophysiological responses one, two, seven and 14 days following acoustic trauma by low-frequency tones. Previously established relationships between injury and response potentials are confirmed for the areas of major injury. These relationships do not exist, however, in other areas showing little or no injury. Healing of lesions in the organ of Corti occurs largely in the first week after injury and is essentially complete in two weeks. Recovery of injured cells to normal appears to follow a similar course when it occurs. Further degeneration of injured cells may continue for much longer periods.

Several combinations of specific injuries to the organ of Corti and the basilar membrane are described, and their implications with respect to function and the recovery process are discussed.

REFERENCES.

1. Covell, W. P. and Eldredge, D. H.: Injury to Animal Ears by Intense Sound. AF Technical Report No. 6561, Part I, 1951, Part II, 1952, Wright Air Development Center, Ohio.

2. Davis, H. et al.: Acoustic Trauma in the Guinea Pig. *Jour. Acoust. Soc. Am.*, 25:1180-1189, 1952.

3. Schuknecht, H. F., and Neff, W. D.: Hearing Losses After Apical Lesions in the Cochlea. *Acta Otol.-Laryngol.*, 42:263-274, 1953.

4. Schuknecht, H. F., Techniques for the Study of Cochlear Function and Pathology in Experimental Animals: *Arch. Otolaryngol.*, 58:377-397, 1953.

5. Davis, H., Gernandt, B. E., Riesco-MacClure, J. S., and Covell, W.

P.: Aural Microphonics in the Cochlea of the Guinea Pig. *Jour. Acoust. Soc. Am.*, 21:502-510, 1949.

6. Tasaki, I., Davis, H., and Legouix, J. P.: The Space-Time Pattern of the Cochlear Microphonics (Guinea Pig) as Recorded by Differential Electrodes. *Jour. Acoust. Soc. Am.*, 24:502-519, 1952.

7. Covell, W. P.: Histologic Changes in the Organ of Corti with Intense Sound. *Jour. Comp. Neur.*, 99:43-60, 1953.

8. Benson, R. W.: The Calibration and Use of Probe-Tube Microphones. *Jour. Acoust. Soc. Am.*, 25:128-134, 1953.

9. Alexander, I. E., and Githler, F. J.: Histological Examination of Cochlear Structure Following Exposure to Jet Engine Noise. *Jour. Comp. and Physiol. Psychol.*, 44:513-524, 1951.

10. Smith, C. A., Lowry, O. H., and Wu, M-L.: The Electrolytes of the Labyrinthine Fluids. The Laryngoscope, 64:141-153, 1954.

11. v. Békésy, G.: DC Resting Potentials Inside the Cochlear Partition. *Jour. Acoust. Soc. Am.*, 24:72-76, 1952.

12. Tasaki, I., Davis, H., and Eldredge, D. H.: Exploration of Cochlear Potentials in Guinea Pig with a Microelectrode. *Jour. Acoust. Soc. Am.*, 26:765-773, 1954.

13. Schuknecht, H. F.: Presbycusis. The Laryngoscope, 65:402-419, 1955.

14. Covell, W. P., and Rogers, J. B.: Pathologic Changes in the Inner Ear of Senile Animals (Guinea Pig). (Manuscript in preparation).

15. Riskaer, N., Christensen, E., Petersen, P. V., and Weidman, H.: The Ototoxicity of Neomycin. *Acta Oto-Laryngol.*, 46:137-152, 1956.

16. Covell, W. P.: The Lesions of Experimental Acoustic Trauma. *Abstr., VIe Congres Federatif International D'Anatomie*, Paris, 25-30 July, 1955, Masson & Cie, p. 53.

17. v. Békésy, G.: Description of Some Mechanical Properties of the Organ of Corti. *Jour. Acoust. Soc. Am.*, 25:770-785, 1953.

18. Katsuki, Y., and Covell, W. P.: The Organ of Corti by Phase Contrast Microscopy. The Laryngoscope, 63:1-17, 1953.

19. Schnuknecht, H. F.: Lesions of the Organ of Corti. *Trans. Am. Acad. Ophthal. and Otolaryngol.*, 366-383, 1953.

INTERNATIONAL VOICE CONFERENCE.

Following the International Congress of Otolaryngology in Washington, D. C., next Spring, there will be an International Voice Conference (Laryngeal Research, Function and Therapy) in Chicago, Illinois, May 20-22, 1957. For Information address: Dr. Hans von Leden, 30 North Michigan, Chicago 2, Ill., U.S.A.

METASTATIC MULTIPLE MYELOMA TO THE LARYNX.

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INTRODUCTION.

Metastasis of malignant disease to the larynx is not a common occurrence due to the anatomical construction of the larynx, its blood supply and lymphatic drainage. The majority of the cases of metastatic disease reported have been of patients with malignant melanoma. While there have been numerous reports of plasma cell myelomas of the upper respiratory tract involving the larynx, (Havens[1], Priest[2], Hellwig[3], Vogt[4], Walther[5], and others), cases of multiple myeloma with metastasis of the larynx have been rare. Individual cases have been reported by Pearson[6], Verebely[7], and Gillmore[8]. Because of the paucity of cases reported, it is felt that this case of malignant myeloma with metastasis to the larynx should be recorded.

CASE HISTORY.

L. W. S., a 49-year-old white male, was first seen January 8, 1954, in acute distress from laryngeal obstruction. An immediate tracheotomy was performed. Following this, his history was obtained and a physical examination completed. Approximately two weeks prior to his admission, the patient noticed a hoarseness which grew progressively worse, and one week later he developed increasing respiratory stridor. The patient complained of persistent gastritis, cough and weight loss for about 18 months to two years. During the past year he had developed increasing pain in his left hip and back. In November, 1953, the patient fell and injured his left hip, which had become progressively more disabling. For the past month he also had chest and back pains.

The physical examination showed an emaciated white male, who appeared to be chronically ill. The findings were as follows:

Eyes—Pupils react to light and accommodations. E. O. M., fields and conjunction negative. Fundi demonstrated physiologic cupping.

Nose—Septum deviated to left, otherwise findings were negative.

Ears and Mouth—Findings were within normal limits.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, Sept. 25, 1956.

Larynx—A large fungating lesion was found protruding through the vocal cords from the left subglottid area.

Neck—No palpable, firm lymph nodes. Tracheotomy tube was in place.

Chest—Except for a tenderness over the eighth and ninth ribs, the findings were essentially negative.

Heart—The findings were essentially negative.

Abdomen—Soft; no masses, no tenderness.

Extremities—Findings were negative except for tenderness on movement of the left hip.

The initial impression, based upon the physical findings, was carcinoma of the larynx with metastasis to the chest and hip. The biopsy of the larynx was reported as plasma cell myeloma. An X-ray survey of the chest showed fractures of the right ninth rib in the midaxillary line, and of the left eighth rib in the axillary line, with previous fractures of the right eleventh and left tenth ribs. X-ray studies of the lumbar spine were not significant except for moderate osteoarthritic changes. An oval area of radiolucenty was seen in the left femur at the level of the intertrochanteric line, with multiple scattered smaller areas in the pelvis and right femur. These areas had the appearance of metastatic lesions. The routine studies were as follows: Urinalysis—Negative with the exception of a three plus albumin. The Bence-Jones protein was positive in four of five examinations. The blood count revealed 4.3 million erythrocytes, 12.5 gm. hemoglobin, 6,900 leukocytes with 81 per cent polymorphonuclear cells, 14 per cent lymphocytes, 4 per cent monocytes and 1 per cent eosinophiles. This led to an obvious diagnosis of multiple myeloma with metastasis to the larynx.

The patient was placed on Urethane, 2 gm. twice daily. His improvement was rapid. Within ten days he had no pain in his chest or hip, and on examination of the larynx, no lesion could be found. The patient experienced no nausea from the Urethane, but there was a steady drop in his white blood count. He was discharged from the hospital January 30, 1954, greatly improved. Urethane was continued. The tracheotomy tube was closed but left in position. The patient was instructed to return for frequent blood studies.

He progressed satisfactorily until April 7, 1954, when the leukocyte count was found to be 2,860. The Urethane was immediately discontinued. Almost simultaneously the patient began to regress with increasing hip pain and progressive shortness of breath. He was readmitted to the hospital April 12, 1954. On readmission, the patient had a recurrence of the lesion in the left subglottid area with two firm fixed masses 3 cm. in diameter on either side of the trachea, just inferior to the tracheotomy opening. With this exception the physical findings were essentially the same as on the previous admission. The leukocyte count remained at 2,800, with an erythrocyte count of 3.1 million and 10 gm. of hemoglobin.

Therapy was begun with small doses of X-ray to the larynx and metastatic cervical nodes. Repeated blood transfusions were given and ACTH, 50 mgm. day, was instigated May 5, 1954. The patient's pain became more severe, the respiratory symptoms persisted, but the leukocytes increased. The white blood count rose to 6,100 by May 13, 1954, and the Urethane therapy was repeated. A soft tissue nodule measuring 1.5x1.5 cm. developed on the left chest wall at the eighth

rib. The patient slowly improved on Urethane and was again discharged May 29, 1954, although pain persisted in the left hip.

Two weeks later, however, the patient's leukocyte count again fell to dangerous levels. He then had a steady down hill course, which terminated in six weeks.

DISCUSSION.

This case is of interest because it is one of the first reported cases of multiple myeloma with metastasis to the larynx, and because of the rapid response to Urethane. It is also of interest to note that the pain increased and a soft tissue mass developed during ACTH therapy, although the leukocyte count rapidly increased.

SUMMARY.

A case of multiple myeloma with metastasis to the lorynx has been reported, along with the patient's response to Urethane and ACTH.

REFERENCES.

1. HAVENS, F. Z., PARKHILL, E. M.: Tumors of the Larynx Other than Squamous Cell Epithelomas. *Arch. Otolaryn.*, 34:1113-1122,1941.

2. PRIEST, R. E.: Extra-Medullary Plasma Cell Tumors of the Nose, Pharynx and Larynx. *Trans. Amer. Laryngol., Rhinol. and Otol.*, 278-287, 1952.

3. HELLWIG, C. A.: Extra-Medullary Plasma Cell Tumors as Observed in Various Locations. *Arch. Path.*, 36:96-111, 1943.

4. VOGT, S.: Mentioned in Reference 8.

5. WALTHER, H.: Mentioned in Reference 8.

6. PEARSON, B., STARK, E., and KEPL, M.: Multiple Myeloma with Laryngeal Involvement. *Arch. Path.*, 36:321, 1943.

7. VEREBELY: Mentioned in Reference 6.

8. GILMORE, G. B.: Multiple Myeeloma with Laryngeal Metastasis. *Arch. Otolaryngol.*, 34:453-460, 1941.

GLOMUS JUGULARIS TUMOR

(Nonchromaffin Paraganglion of Middle Ear).

ROBERTO MARINHO, M.D., and ERNANI T. TORRES, M.D.,**

Rio de Janeiro, Brazil.

The glomus jugularis are nonchromaffin paraganglions described by Guild[1] in 1941 and found in the adventitia of the jugular bulb and along the nerve of Jacobson. The same author[2] in 1953, described a similar body found along the nerve of Arnold, and both descriptions allowed for the diagnosis of certain tumors of the middle ear arising from these vascular bodies.

Histologically the glomus jugularis are identical to the carotid bodies, and are composed of a ball of winding capillaries separated by epithelioid-like cells and a connective collagenous estroma. The main cells are uniform in size and shape, rich in cytoplasm, and the nuclei are round or oval and regular in size. These small structures are also found in the mediastinum and neck, always in relation to blood vessels and nerves.

Rosenwasser[3] in 1945 reported the first case of a tumor of the glomus jugularis, and other authors later published cases of identical tumors totaling 42 cases up to 1951, according to Winship and Louzan[4], and totaling 101 cases until 1954, according to Dias and Figueiredo[5], including the reviewed cases originally reported with diagnosis of angiomatous tumor type of the middle ear. Other cases were reported later, and this increase is probably due to publications on the subject making diagnosis easier and thus suggesting that the incidence of these tumors is not so rare as was first believed.

REPORT OF A CASE.

In July, 1955, a 35-year-old white man was admitted to the Casa de Saude São Miguel, complaining of copious sanguine-purulent exudate

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This ms. received in the Laryngoscope Office and accepted for publication July 26, 1956.

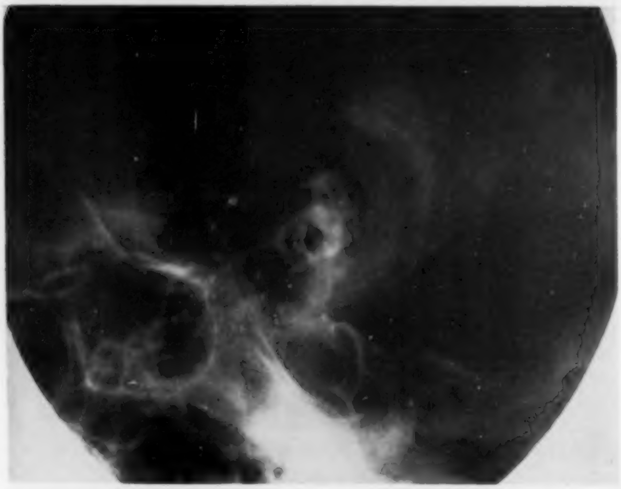


Fig. 1. Schuller's Projection.

Fig. 2. Stenvers' Projection.

in the left ear for the past three months, later paralysis of the left facial nerve and tumefaction of the parotid gland.

Ear examination showed a large cavity similar to that observed after recent radical mastoidectomy, with ample communication to the attic and antrum and extensive erosion of the external ear canal. There was communication with the glenoid cavity, the condyle of the mandible bone was destroyed and no evidence of growth was found. The tympanic membrane was completely damaged and the tegmen intact. Conductive hearing loss on the left ear and no clinical symptoms of involvement of the vestibular labyrinth or other cranial nerves. Otherwise physical examination was normal.

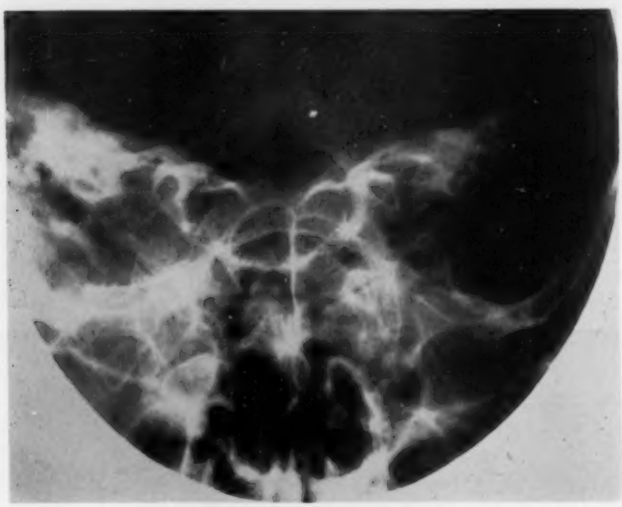
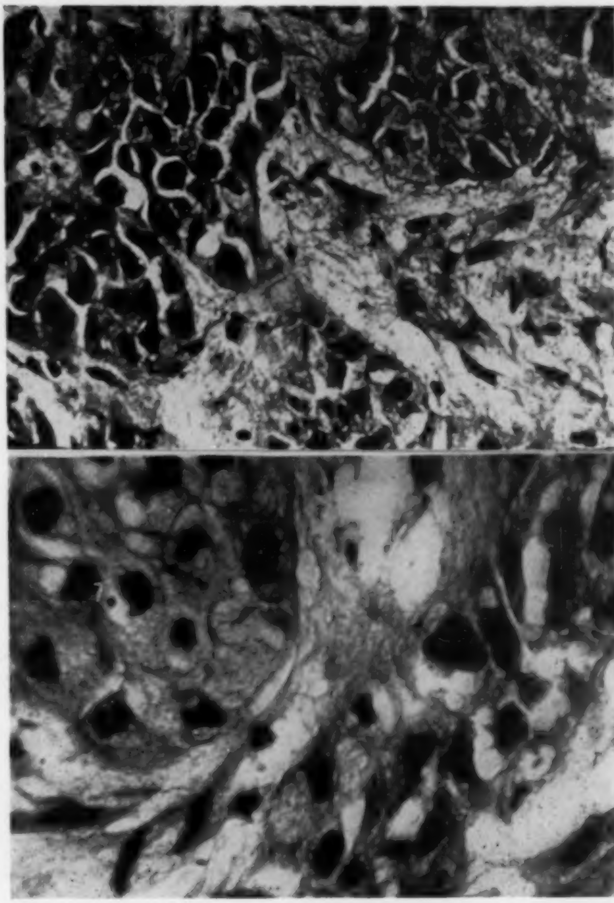


Fig. 3. Worms' Projection.

The bacteriological tests revealed pyocyanic bacillus and the cytological examination, chronic inflammatory process. Sialography of the parotid gland was normal and X-rays in Schuller's (see Fig. 1); Mayer's, Stenvers' (see Fig. 2); Worms' (see Fig. 3), and Hirtz's projections, revealed ample area of osteolysis involving the whole temporal squamous, the annulus tympanicus, the zygomatic root, the glenoid cavity and the condyle of the mandible bone, as well as the base of the petrous bone to the bony labyrinth.

Polymixin alternating with dihydrostreptomycin was prescribed, decreasing the secretion.

Following the first visit unsuccessful efforts were made to remove fragments for histological examination. Failure of the biopsy was due to the existence of necrotic tissue and extensive bleeding, which occurred



Figs. 4 and 5. Glomus Jugularis Tumor. H.E. x260 and x380.

when attempts were made to remove small granulations from the hypotympanum.

Some five weeks after the admission of the patient to the Hospital a small tumor was first seen in the posterior region of the external ear canal. A biopsy was made and the diagnosis established.

Histopathological Report: The biopsy specimen measured 5x3x2 mm., very friable and dark red in color. Microscopically the tumor was

composed of groups and whorls of polygonal cells in a vascular estroma. A fine honey-comb network of reticular fibers was observed in the septa but not in the cells. The tumor cells were very irregular in size and shape with large nuclei, a few anormal mitotic figures were seen, and the vessels sometimes were lined by tumor cells (see Fig. 4 and 5).

Histopathological diagnosis: Glomus jugularis tumor.

The patient was obviously inoperable from the first examination. Although we knew from the existent otologic literature that such tumors are not radiosensitive, a tentative palliation with Roentgen therapy was made, with negative results. The patient died about four months after his admission to the Hospital with evident symptoms of brain involvement.

SUMMARY.

At the first examination patient showed extensive erosion of the temporal bone but no evidence of tumor. Five weeks later, after several unsuccessful attempts at biopsy, a tumor was first seen and histological diagnosis established.

The lesion was so extensive that surgery was inadvisable and Roentgen therapy was unsuccessful.

Death occurred four months after the admission of the patient to the Hospital with evident symptoms of brain involvement.

REFERENCES

1. Guild, S. R.: A Hitherto Unrecognized Structure, the Glomus Jugularis in Man. *Anat. Rec.* (Supp. 2), 79:28, Mar., 1941.

2. Guild, S. R.: The Glomus Jugulare, a Nonchromaffin Praganglion in Man. *Ann. Otol., Rhinol. and Laryngol.*, 62:1045, Dec. 1953.

3. Rosenwasser, H.: Carotid Body Tumor of the Middle Ear and Mastoid. *Arch. Otalaryngol.*, 41:64, Jan., 1945.

4. Winship, T., and Louzan, J.: Tumors of the Glomus Jugulare Not Associated with the Jugular Vein. *Arch, Otolaryngol.*, 54:378, Oct., 1951.

5. Dias, M. C. S., and Figueiredo, M. A.: Paraganglioma Acromafinico do Ouvido Medio. *Revista Brasileira de Oto-rino-laringologia*, 22:335, Nov.-Dec., 1954.

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A SIMPLE METHOD TO REDUCE BLEEDING DURING TONSILLECTOMY.*

CAESAR N. ABU-JAUDEH, M.D.,

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Although never serious, bleeding during tonsillectomy may be quite annoying. There are at least three good reasons for the desire to reduce bleeding to a minimum during tonsillectomy:

1. It is the aim of every surgeon to lose as little blood as possible at operation.

2. When bleeding is excessive, there is inadequate visual control. Haste on the part of the surgeon to complete the operation might result in injury to the tonsillar pillars, especially the posterior pair; also the surgical capsule is not hugged during the dissection, and the peritonsillar venous plexus is injured, resulting in more bleeding.

3. There is a greater risk of blood aspiration into the tracheo-bronchial tree.

The amount of blood loss during tonsillectomy under local anesthesia is surprisingly small compared to the amount lost when a general anesthetic is used. This difference is obviously due to the local injection of procaine-epinephrine into and around the tonsil. It was hoped to achieve similar results when such an injection was given for tonsillectomies performed under general anesthesia. Instead of procaine, however, normal saline was used as the former was not necessary.

Injection of saline-epinephrine is frequently used in connection with post-auricular and endaural mastoid incisions and gingival incisions for the Caldwell-Luc procedure, and its efficacy as a hemostat is universally acknowledged.

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Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication, January 9, 1956.

Moore,[1] reporting recently on the use of saline-epinephrine peritonsillar injection for tonsillectomies under general anesthesia, stated that the results have been most gratifying. He has been using this method routinely on children and adults for several years.

The object of this article is to evaluate the effect of local saline-epinephrine injection on the amount of blood loss during tonsillectomy when the latter is performed under general anesthesia.

METHOD USED TO COMPUTE BLOOD LOSS DURING TONSILLECTOMY.

The standard tonsil sponge used by us during tonsillectomy for swabbing measures 2"x12" and is four-ply. The quantity of blood necessary to saturate such a sponge is approximately 10 cc. The sponges are counted at the end of the operation, and the quantity of blood loss computed. Adenoidectomy is performed first, the nasopharynx packed off and the throat cleaned from blood, mucus and saliva. Sponges used for this part of the operation are discarded into a separate bucket, and only the sponges used for the tonsillectomy are counted.

OPERATIVE PROCEDURE.

The solution used to inject the tonsils is prepared by adding 10 drops of 1-1000 epinephrine solution to 30 cc. of normal saline. About 7 to 10 cc. of this solution is injected into and around each tonsil in the same manner as that used for the injection of procaine in the local tonsil operation. The tonsils are then removed by the usual Crowe dissection technique.

RESULTS.

One hundred cases (children and adults), upon whom adenotonsillectomy or tonsillectomy alone was done during 1952-1955 are presented in this report. Fifty cases, selected at random, received the saline-epinephrine injection and the other 50 were used as controls. The same preoperative medication, preparation and operative technique were used in all patients except for the saline-epinephrine injection.

In the group receiving the preoperative local injection, the

maximum blood loss was 40 cc., and the minimum was 10 cc. The average blood loss was 20 cc. In the second group (those not receiving the injection) the maximum blood loss was 170 cc., and the minimum was 100 cc. The average blood loss was 120 cc.

In none of the 100 cases operated upon was there primary bleeding. One patient from the control group had secondary bleeding five days after operation.

SUMMARY AND CONCLUSIONS.

In a series of 100 tonsillectomies done under general anesthesia, the average blood loss for those receiving a saline-epinephrine injection was 20 cc. The average blood loss for those not receiving the injection was 120 cc. This difference is significant.

Although the injection prolongs the operation by a few minutes, its hemostatic effect is marked. It allows leisurely dissection of the tonsils and greatly reduces the possibility of injury to the tonsillar pillars and peritonsillar venous plexus and aspiration of blood into the lower respiratory passages. Its adoption is highly recommended.

REFERENCE.

1. MOORE, P. M.: General Tonsillectomy. *Ann. Otol., Rhinol., and Laryngol.*, 64:494, 1955.

American University Hospital.

UNIVERSITY OF CINCINNATI

The Twenty-First Annual Graduate Convention in Otology, Rhinology and Laryngology will be held at the University of Cincinnati College of Medicine, April 15-20, 1957. An intensive course will be given in Regional Anatomy and Cadaver Surgery; also special lectures. Excellent anatomical material will be available. For additional information contact Dr. Henry M. Goodyear, 19 Garfield Place, Cincinnati 2, Ohio.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The meeting dates of the Sixth International Congress of Otolaryngology are again emphasized as May 5th through May 10th, 1957. The scientific program for the Plenary Sessions is now complete and is as follows:

CHRONIC SUPPURATION OF THE TEMPORAL BONE.

OPENERS: Marcus Diamant, Central County Hospital, Halmstad, Sweden—Anatomical Etiological Factors in Chronic Middle Ear Discharge.

Luzius Rüedi, Zurich, Switzerland—Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone.

Horst Wullstein, Director, Otolaryngological Clinic, University of Würzburg, Germany—Surgical Repair for Improvement of Hearing in Chronic Otitis Media.

DISCUSSERS: A. Tumarkin, Liverpool, England; Juan Manuel Tato, Buenos Aires, Argentina; T. E. Cawthorne, London, England; Fritz Zöllner, Freiburg, Germany.

COLLAGEN DISORDERS OF THE RESPIRATORY TRACT.

OPENERS: Hans Selye, Director, Institute of Experimental Medicine and Surgery, University of Montreal, Faculty of Medicine, Montreal, Canada.

Introduction:

Michele Arslan, Padua, Italy—The Upper Respiratory Tract.

Leslie Gay, Physician-in-Charge, Allergy Clinic, The Johns Hopkins Hospital, Baltimore, U. S. A.—The Lower Respiratory Tract.

DISCUSSERS: Victor E. Negus, London, England; Branimir Gusic, Zagreb, Yugoslavia; Aubrey G. Rawlins, San Francisco, U. S. A.; Henry L. Williams, Rochester, Minn., U. S. A.

PAPILLOMA OF THE LARYNX.

OPENERS: Jo Ono, Tokyo, Japan—Etiology.

Plinio de Mattos Barretto, Faculty of Medicine, University of Sao Paulo, Brazil.

Diagnosis:

F. C. W. Capps, London, England—Therapy.

DISCUSSERS: C. A. Hamberger, Göteborg, Sweden; Pedro Hernandez Gonzalo, Havana, Cuba; Eelco Huizinga, Groningen, Netherlands; Albert von Riccabona, Vienna, Austria.

Anyone planning to attend the Congress and who has not yet registered should do so immediately in order to obtain hotel registration priority.

For more detailed information pertaining to the Sixth International Congress please communicate with the General Secretary, 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

MOUNT SINAI HOSPITAL SPECIAL COURSE.

A special course in Rhinoplasty and Reconstructive Surgery of the Septum, following the Sixth International Congress of Otolaryngology, May 13, 1957, to May 18, 1957, will be given at the Mount Sinai Hospital, New York under the direction of Dr. Irving B. Goldman. This will be open only to foreign colleagues on a full scholarship basis.

An intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given July 13, 1957, to July 27, 1957, by Dr. Irving B. Goldman and staff at the Mount Sinai Hospital, New York in affiliation with Columbia University.

Candidates for either course should apply to Registrar for Post-graduate Medical Instruction, the Mount Sinai Hospital, Fifth Avenue and One-hundredth street, New York 29, New York.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Ill. ....
Vice-President: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Washington, D. C., May 4, 1957.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

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First Vice-President: Dr. Henry M. Goodyear, Cincinnati, Ohio.
Second Vice-President: Dr. Robert E. Priest, Minneapolis, Minn.
Secretary: Dr. Harry P. Schenck, Philadelphia, Pa.
Treasurer: Dr. Fred W. Dixon, Cleveland, Ohio.
Meeting: Statler Hotel, Washington, D. C., May 3, 1957.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

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President-Elect: Dr. Lewis F. Morrison.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Statler Hotel, Washington, D. C., May, 1957.

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Secretary: Hugh A. Kuhn, M.D., Hammond, Ind.
Representative to Scientific Exhibit: Walter Heck, M.D., San Francisco, Calif.
Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 13-19, 1957.

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Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 21-23, 1958.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October 6-12, 1957.

THE AMERICAN RHINOLOGIC SOCIETY.

President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. James Chessen, 1829 High St., Denver, Colo.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, Illinois, October, 1956.
Annual Meeting: Palmer House, Chicago, Illinois, October, 1957.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. D. M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1957.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

President: Dr. Irvin J. Fine, 506 New Brunswick Ave., Perth Amboy, N. J.
Secretary: Dr. William Schwartz, 224 Lexington Ave., Passaic, N. J.
Meetings: Quarterly.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Gordon D. Hoople, 1100 East Genesee St., Syracure, N. Y.
Secretary: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1957.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Arnold L. Caron, 36 Pleasant St., Worchester, Mass.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Jose Gros, Havana, Cuba.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Sixth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Brazil, 1958.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago 11, Ill.
Meeting: Statler Hotel, Washington, D. C., May 5 - 10, 1957.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

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Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
Meeting: First Monday of each Month, October through May.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

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Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Virginia.
Meeting: Roanoke, Virginia, December 6 and 7, 1957.

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Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

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Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: The Edgewater Gulf Hotel, Edgewater Park, Miss., May 17-18, 1957.

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Meeting:

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Time: 6:00 P.M., first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P. M. first Thursday of each month from September to June inclusive—Ophthalmology Section.

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